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TONSILLECTOMY

LOCAL RESULTS AND INFLUENCE OF THE OPERATION ON SURROUNDING TISSUES

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PHILADELPHIA

The problem of the recurrence of tonsillar tissue following tonsillectomy is one that has received much consideration. In spite of many articles drawing attention to the local results of this operation and investigations to reveal the causes of the recurrence of tonsillar tissue, tonsillectomized patients are constantly returning to those who operated on them, not only unimproved symptomatically but with throats showing the signs of incomplete and poorly performed operations. It is true that the surgical work on many of them has been performed by general practitioners, general surgeons and at times pediatricians, but on many others it has been done by specialists in otolaryngology. It is my purpose in this presentation to reveal the results of a statistical study of tonsillectomized persons as regards the recurrence of tonsillar tissue, injury to the faucial pillars and hypertrophy of pharyngeal lymphoid tissue and to draw a comparison between results obtained by practitioners and those obtained by specialists. No attempt will be made to evaluate the results from the symptomatic standpoint, and the usual review of the anatomy and the theories of the physiology of the tonsils will be omitted.

METHOD OF INVESTIGATION

During the years of 1936, 1937 and 1938 I examined 887 girls entering the freshman class at the University of Pennsylvania, in the Student Health Department. Their ages ranged from 18 to 21. The examination of the throat consisted in careful inspection of the tonsils or the tonsillar fauces (with the tongue well depressed and with the use of the pillar retractor when necessary), the posterior pharyngeal wall, the palate and the faucial pillars. With those on whom tonsillectomy had been performed, 621 in number, note was made whether or not lymphoid tissue was present in the tonsillar fauces. If so, the amount

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and character of this tissue were observed and recorded. In 153 of the tonsillectomized students, in addition to a study of the tonsillar fauces, the amount of scarring of the tonsillar pillars, fauces and palate and the amount of pharyngeal lymphoid tissue were noted. With the latter group it was also recorded whether the operation had been performed by the family doctor or by a specialist.

When remnants of tonsils were found, they were classified as infected or noninfected. If the remnants appeared to be simply non-inflamed lymphoid tissue, without observable crypts containing exudate or without signs of being partially obscured by scar tissue, they were classified as noninfected. If the remnants showed signs of chronic inflammation, or if there were crypts containing exudative material, or if they were embedded and partially covered by fibrous scar, they were classified as infected.

A small amount of lymphoid tissue in the pharynx was not considered abnormal, but an excessive amount was recorded. A comparison was drawn between the condition of the throat in which the operation had been performed by a specialist and the throat in which the tonsillectomy had been performed by the family doctor. Minor scarring of the pillars and fossae was not recorded, but only such conditions as asymmetry of the palatal arch, distortion of the palate by fibrosis, loss of portions of the pillars or considerable fibrous scar in the tonsil fossa.

RESULTS

The accompanying tables give an outline of the results obtained from this investigation. Only 30 per cent of the 887 students examined had not had their tonsils removed. Of the 621 students who had been operated on, 77.3 per cent showed tonsillar remnants in the fossae. Of these, 66.6 per cent were placed in the class of noninfected remnants and 10.6 per cent in the class of infected remnants. In the group of 153 students who underwent more detailed investigation 54.9 per cent were found to have an excessive amount of lymphoid tissue in the pharynx and 22.8 per cent showed scarring of the palate, pillars or fossae. Of these 153 patients 67.3 per cent had been operated on by otolaryngologists and 28.7 by family physicians. Of the 103 students who had been operated on by specialists 74.7 per cent showed tonsillar remnants, 14.5 per cent showed scars and 25.3 per cent had no lymphoid tissue in the tonsillar fossae. Of the 44 students operated on by family physicians 79.5 per cent showed tonsillar remnants, 40.9 per cent showed scars and 20.5 per cent had fossae free from lymphoid tissue. One hundred per cent of the 21 students who had been operated on before the fourth year of age showed remnants of tonsillar tissue in the fossae; 73.4 per cent of the 94 students who had been operated on between the fourth and the tenth year of age showed lymphoid tissue

in the fossae, and 96.1 per cent of 26 students operated on after the tenth year of age showed recurrence of tonsillar tissue in the fossae.

STATISTICS AND OPINIONS ON THE RECURRENCE OF TONSILLAR TISSUE OBTAINED FROM THE LITERATURE

In 1923 Grichter and Pearlman [1] found that in 25 per cent of their cases lymphoid structure was demonstrable in the tissues taken from the margins of the tonsillar bed recently operated on.

Baum [2] stated that the recurrence of lymphoid tissue in the tonsillar fossae is not due entirely to faulty technic, as there is extracapsular lymphoid tissue in the tonsillar fossae, in the pillars, in the plicae and between the base of the tongue and the tonsil.

Others—such as Spake,[3] McMurray,[4] Long,[5] Roberts [6] and Pierce [7]—have noted the recurrence of tonsillar tissue in various parts of the fossae after apparently complete enucleation.

Leshin and Pearlman [8] examined microscopically 155 sections of the plica triangularis, plica supratonsillaris and plica retrotonsillaris and found lymphoid tissue in practically 50 per cent of these sections.

In a study of the end results of operations on tonsils and adenoids in children Nesbit [9] examined 1,138 persons operated on. There was noted scarring of the faucial pillars in 753. The palate was scarred and drawn tightly across the fauces in 3. The uvula had been removed in 7. Distinct lymphoid nodules in the postpharyngeal wall were present in 463, and the lymphoid tissue in the lateral pharyngeal bands was hypertrophied in 473. The whole picture showed the tendency to compensatory hypertrophy of the lymphoid tissue in the oropharynx.

Epstein [10] made careful observations on 540 children between the ages of 2 and 13 years before and at intervals for two years after

1. Grichter, B. B., and Pearlman, S. J.: Lymphoid Tissue in the Tonsillar Fossae Following Tonsillectomy, Ann. Otol., Rhin. & Laryng. **32**:860, 1923.

2. Baum, H. L.: Complications of Tonsil and Adenoid Operations: Their Prevention and Management, Ann. Otol., Rhin. & Laryng. **28**:37, 1919.

3. Spake, L. B.: The Recurring Tonsil, J. Kansas M. Soc. **22**:321, 1922.

4. McMurray, J. B.: A Study of the Superficial and Deep Lymphoid Tissues of the Hypopharynx, Atlantic M. J. **30**:63, 1926.

5. Long, C. H.: Recurrence of Tonsils After Tonsillectomy, Illinois M. J. **45**:226, 1924.

6. Roberts, E. R.: The Tonsil Problem, Arch. Otolaryng. **6**:565 (Dec.) 1927.

7. Pierce, N.: The Tonsil Problem, Tr. Am. Laryng., Rhin. & Otol. Soc. **48**:267, 1921.

8. Leshin, N., and Pearlman, S. J.: Are Tonsillar Recurrences Entirely Due to Faulty Operative Technic? Arch. Otolaryng. **13**:37 (Jan.) 1931.

9. Nesbit, E.: The Post-Operative Complications and End Results of Tonsil and Adenoid Operations in Children, Brit. M. J. **2**:509, 1934.

10. Epstein, M. E.: Factors Influencing the Results of Tonsillectomy and Adenoidectomy, Am. J. Dis. Child. **53**:1503 (June) 1937.

TABLE 1.—*Results of the Examination of the Girl Students at the University of Pennsylvania*

Year Examination Was Made	Number of Girls Examined	Age Range at Time of Examination	Number Who Had Not Had Tonsillectomy	Number Who Had Had Tonsillectomy	Number Operated on Who Had Noninfected Remnants	Number Operated on Who Had Infected Remnants	Number Operated on Whose Tonsillar Fossae Were Free from Tonsillar Tissue
1936	430	18 to 21	144	286	192	22	69
1937	241	18 to 21	59	182	124	29	26
1938	216	18 to 21	63	153	98	15	46
Total for the three years	887		266	621	414	66	141

TABLE 2.—*Results of Detailed Study of One Hundred and Fifty-Three Girl Students Examined in 1938 Who Had Undergone Tonsillectomy*

Number operated on before the fourth year of age	21
Number operated on between the fourth and the tenth year of age	94
Number operated on after the tenth year of age	26
Number operated on twice	7
Number operated on at an age not ascertained	8
Number operated on by specialists (otolaryngologists)	103
Number operated on by family physicians	44
Number operated on showing scarring of palate, pillars or fossae	35
Number operated on showing an excessive amount of lymphoid tissue in the pharynx	84
Number operated on by specialists showing remnants	77
Number operated on by specialists showing clean fossae	26
Number operated on by specialists showing scars	15
Number operated on by family physicians showing remnants	35
Number operated on by family physicians showing clean fossae	9
Number operated on by family physicians showing scars	18
Number operated on before the fourth year of age showing remnants	21
Number operated on between the fourth and the tenth year showing remnants	69
Number operated on after the tenth year of age showing remnants	23

TABLE 3.—*Summary of Results of Study in Percentages*

Percentage of 887 students examined who had not had tonsillectomy	30.0
Percentage of 887 students examined who had had tonsillectomy	70.0
Percentage of those operated on (621) whose fossae showed remnants	77.3
Percentage of those operated on (621) whose fossae showed noninfected remnants	66.6
Percentage of those operated on (621) whose fossae showed infected remnants	10.6
Percentage of those operated on (621) whose fossae showed no remnants	22.7
Percentage of 153 students operated on who showed an excessive amount of lymphoid tissue in the pharynx	54.9
Percentage of 153 students operated on who showed scarring of the palate, pillars or fossae	22.8
Percentage of 153 students operated on by specialists (otolaryngologists)	67.3
Percentage of 153 students operated on by family physicians	28.7
Percentage of 103 students operated on by specialists showing tonsillar remnants	74.7
Percentage of 106 students operated on by specialists showing fossae free from tonsillar tissue	25.3
Percentage of 103 students operated on by specialists showing scars	14.5
Percentage of 44 students operated on by family physicians showing tonsillar remnants	79.5
Percentage of 44 students operated on by family physicians showing fossae free from tonsillar tissue	20.5
Percentage of 44 students operated on by family physicians showing scars	40.9
Percentage of 21 students operated on before fourth year of age who showed tonsillar remnants	100.0
Percentage of 94 students operated on between the fourth and the tenth year of age who showed tonsillar remnants	73.4
Percentage of 26 students operated on after the tenth year of age who showed tonsillar remnants	96.1

tonsillectomy and adenoidectomy. Of these, 336 showed hypertrophied lymphoid tissue. This tendency to hypertrophy was especially evident in children under 4 years of age and least evident in those over 10 years of age.

Miller [11] reported tonsillar tissue, in varying amounts, that had been left unknowingly or that had regenerated in 21.8 per cent of 133 tonsillectomized patients. In all but 2 subjects the tissue was at the base or near the base of the fossa.

Regarding the incidence of tonsillectomy, Glover [12] stated that it has been estimated that the number of operations of this type forms one third of the number of operations in general performed with patients under general anesthesia in the United States.

Cunningham [13] found that one third of 12,530 young white women who entered the University of California as students between 1920 and 1929 had had an operation for removal of tonsils.

Paton [14] noted that 42.9 per cent of 424 girls between the ages of 13 and 15 years who were examined on admittance to a boarding school had had their tonsils removed.

Clark [15] examined 143 patients who had been subjected to so-called tonsillectomy and found that 59 of this number had tonsillar stumps.

In a study of 167 operations for removal of tonsils and adenoids Bradley [16] reported that in only 18 per cent was eradication complete. He quoted Coues as having found 30 per cent of tonsillectomized children suffering from chronic tonsillar hypertrophy.

Rhoads and Dick [17] examined 403 nurses entering training at the Presbyterian and Cook County hospitals and found fairly large pieces of tonsillar tissue remaining in the throats of 73 per cent of those who had had their tonsils removed. These authors made bacterial counts on tonsils and tonsillar stumps and found that the stumps harbored more pathogenic bacteria per gram than tonsils removed for the first time.

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11. Miller, A.: A Study of One Hundred and Thirty-Three Tonsillectomies, M. Rec. **144**:367, 1936.

12. Glover, J. A.: The Incidence of Tonsillectomy in School Children, Proc. Roy. Soc. Med. **31**:1219, 1938.

13. Cunningham, R. L.: Normal, Absent and Pathologic Tonsils in Young Women: A Comparison of Histories, Arch. Int. Med. **47**:513 (April) 1931.

14. Paton, J. H. P.: The Tonsil-Adenoid Operation and Some of Its Results, Quart. J. Med. **22**:107, 1928.

15. Clark, J. P.: Results in a Series of Cases of Tonsillectomy, at the Massachusetts General Hospital, Three to Four Years After Operation, Tr. Am. Laryng. A. **35**:43, 1913.

16. Bradley, W. H.: The Tonsils and Nasopharyngeal Epidemics, Arch. Dis. Childhood **5**:335, 1930.

17. Rhoads, P. S., and Dick, G. F.: Efficacy of Tonsillectomy for the Removal of Focal Infection, J. A. M. A. **91**:1149 (Oct. 20) 1928.

COMMENT

The noteworthy feature of the results obtained in this investigation is the extremely high incidence of recurrence of tonsillar tissue following tonsillectomy. When the tonsillar fossae of only 22.7 per cent of 621 persons who have had their tonsils removed are found free from tonsillar tissue, it cannot but reflect discredit on the operation and the operators. This should emphasize the statement made a few years ago by Holding.[18] He said that general surgeons and general practitioners will continue to perform tonsillectomies until the majority of otolaryngologists justify their specialty by performing better operations. Since the investigations of electrocoagulation of tonsils by Shambaugh, Dougherty and Yonker[19] and more recently by Yonker,[20] otolaryngologists have come to look on electrocoagulation of tonsils as unsuccessful, chiefly because of the failure to eradicate this tissue completely, but unless the figures presented in this paper for complete tonsillectomies can be materially improved the exponents of electrocoagulation will have considerable basis for criticism of surgical methods.

What reasons can be found for the high incidence of recurrence of tonsillar tissue? Probably the most outstanding reason is the inefficiently performed operation—that is, the tonsil is incompletely removed, a remnant being left in the fossa, usually in the lower pole. Why is a remnant left in such a high percentage of cases? The chief factors concerned in this are the following: (1) improper administration of the anesthetic; (2) inadequate illumination of the operative field; (3) unskilful assistance at the operation; (4) imperfect inspection of the fossae. All surgeons who perform tonsillectomy many times eventually reach the conclusion that tonsillectomy is not the easy or minor operation that it is so commonly thought but is dependent for its success on the factors mentioned. Unless the child is in the proper stage of anesthesia, with the tongue relaxed and the gag reflex abolished, it is extremely difficult to make the proper dissection of the tonsil and a careful search for remnants. The observation as to the proper stage of anesthesia applies particularly to the removal of adenoids. It is important also to have strong illumination; otherwise a tonsillar remnant will often be left in the lower pole. Of great importance is proper assistance at the operation. It is my opinion that for a good tonsillectomy more skilful assistance is required than for probably any other operation in the otolaryngologic field. In particular, efficient use of the tongue

18. Holding, A. F.: The Need for Improved Technic in Tonsillectomy, Laryngoscope **45**:458, 1935.

19. Shambaugh, G. E.; Dougherty, C. L., and Yonker, W. J.: Electrocoagulation in the Practice of Otolaryngology, S. Clin. North America **17**:1157, 1932.

20. Yonker, W. J.: Obstacles Encountered in Electrocoagulation of Tonsils, Ann. Otol., Rhin. & Laryng. **43**:117, 1934.

depressor and the suction tip to expose the operative site properly is necessary for good work. Incomplete or careless inspection of the area of operation is the reason for the recurrence. Proper anesthesia, illumination and assistance are the important factors in detecting small pieces of lymphoid tissue that may have escaped the surgical technic.

In addition to the inefficiently performed operation, other factors are concerned in the recurrence of tonsillar tissue. Among these may be mentioned: (1) the possibility of regrowth of lymphoid tissue even though the tonsil has been completely removed; (2) proliferation of a lymph node in the fascia covering the tonsillar fossa; (3) hypertrophy of the lymphoid tissue in one of the plicae which may have been intentionally left; (4) extension of the lingual tonsil into the lower pole of the fossa.

It has been thought at times that the recurrent tonsil may have been a new growth of lymphoid tissue in a fossa in which the tonsil was completely removed. This seems unlikely, and it is probable that a tonsil completely removed with its capsule is never replaced by an actual new growth of lymphoid tissue. Roberts [21] claimed that lymph nodes exist in the fascia which often lines the fossa after tonsillectomy and that these may hypertrophy and partially fill in the fossa with lymphoid tissue. He stated further that a chain of lymph nodes situated behind the posterior pillar occasionally undergoes hyperplasia and shows in the free border and anterior surface of the posterior pillar. Experimental work by Leshin and Pearlman [8] demonstrated the existence of lymphoid tissue in the plica triangularis, plica supratonsillaris and plica retrotonsillaris. In the past it has been the practice of some operators to leave these plicae, particularly the plica triangularis, in doing tonsillectomy. If this practice is followed, there will be a recurrence of tonsillar tissue in the lower part of the fossa in a very large percentage of cases. The extension of lingual tonsillar tissue into the lower pole of the tonsillar fossa following tonsillectomy has been noted commonly. There is a tendency for all lymphoid tissue in the throat to undergo compensatory hypertrophy following tonsillectomy, and the lingual tonsillar hypertrophy is often in the direction of the faucial tonsillar area and may fill up half or more of the lower tonsillar bed.

In the study of the tonsillectomized students herein reported some emphasis was placed on the amount of scarring of the pillars, fossae and palate. It was observed that in those operated on by specialists there was considerably less scarring of these tissues than in those who were operated on by general practitioners. This is only what should be expected and emphasizes again the fact that the well performed tonsillectomy is not a simple operation. Although mild injury of the faucial pillars usually produces no symptoms, more severe injury may

21. Roberts, E. R.: The Tonsil Problem, Arch. Otolaryng. **6**:565 (Dec.) 1927.

cause considerable postoperative trouble. Destruction of the posterior pillar may interfere considerably with the function of the eustachian tube and the proper control of the throat in swallowing. It may also cause retractions of the soft palate, which may result in speech defects, as pointed out by Makuen.[22] Marked injury to the pillars and palate may result in considerable contraction or even atresia of the nasopharynx. Scarring in the tonsillar fossae not only distorts the normal appearance of the throat but may enclose tonsillar remnants and is sometimes the basis for postoperative neuroses.

Such enclosed tonsillar remnants are often a greater menace to the patient both locally and generally than the original tonsils. It is not my intention to give any comparison of symptoms occurring before and after the tonsillectomy. This has been thoroughly reported by many writers. I shall emphasize only the point that recurrence of tonsillar tissue and the infection often present in the lymphoid tissue elsewhere in the throat, which has hypertrophied following the operation, are often responsible for the continuation of the symptoms which necessitated the tonsillectomy and often give more local discomfort and symptoms referable to a focus of infection than were caused by the original tonsil.

CONCLUSION

This presentation offers no new solution of the problem of recurrence of tonsillar tissue. The importance of a thorough technic has been emphasized before. Such a technic is the important factor in the prevention of recurrences. It is impracticable, if not impossible, to remove every particle of lymphoid tissue from the throat and nasopharynx. However, it is possible to remove the faucial tonsil completely with the plicae, the pharyngeal tonsil and the greater part of the macroscopic lymphoid tissue in the nasopharynx, pharynx and hypopharynx. This should be done with the greatest care in order that undue scarring of the tissues will not result. More efficient and careful surgical technic is necessary if otolaryngologists are to avoid the symptomatic recurrence and the possible criticism that comes with the return of tonsillar tissue.

SUMMARY

Tonsillectomized persons are constantly returning to otolaryngologists not only unimproved symptomatically but with throats showing the signs of incomplete and poorly performed operations.

A statistical study has been made to reveal the results of tonsillectomy as shown in the throats of 621 girl students entering a university during the years of 1936, 1937 and 1938.

22. Makuen, G. H.: Relation of the Tonsil Operation to the Soft Palate and Voice, Tr. Am. Laryng. A., 1911, p. 223.

Of these 621 students who had been operated on 77.3 per cent showed tonsillar remnants in the fossae.

A more detailed study of 153 of these tonsillectomized students showed scarring of the palate, pillars or fossae in a large percentage of cases and an excessive amount of lymphoid tissue in the pharynx in the majority.

Statistics on the recurrence of tonsillar tissue obtained from the literature are recorded and certain opinions expressed as to the reason for this recurrence.

Factors influencing the efficiency of tonsillectomy and adenoidectomy are discussed in some detail.

Other factors concerned in the recurrence of tonsillar tissue are briefly discussed and explained.

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DISCUSSION

DR. GEORGE B. WOOD, Philadelphia: Two points in Dr. Campbell's paper are of especial interest to me. One concerns the plica triangularis, that fold of mucous membrane which covers the front of the tonsil. Where its edge merges on to the free surface of the tonsil it is firmly attached but separated from the capsule of the tonsil by loose areolar tissue as it runs forward to fold over the palato-glossal muscle. Immediately posterior to its attachment to the tonsillar surface a deep crypt frequently exists. In his description of the plica Dr. Fetterolf includes all the structures anterior to this crypt. If this description is accepted, the plica contains tonsillar tissue. If, however, the conception of the plica is just the fold of mucous membrane covering the anterior surface of the tonsil, there is no lymphoid tissue except in some instances, in which small superficial lymph follicles may be noted near the inferior pole. If left behind after tonsillectomy, these have the same clinical significance as the lymph follicles on the posterior pharyngeal wall.

The other point is in reference to actual recurrence of tonsillar tissue after tonsillectomy. Lymph follicles may develop in the scar six months to a year after the healing of the tonsillar wound. Again, I attach little significance to their presence as far as focal infection is concerned. Stumps of the tonsil itself left behind after an incomplete tonsillectomy are apt to be covered more or less by scar tissue and possess pockets with narrow mouths which are often a fruitful source of focal infection. As a rule it is not difficult to differentiate between these two types of tonssillar tissue in a throat that has been operated on.

NURSING, FEEDING AND PARENTERAL ADMINISTRATION OF FLUIDS

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In attempting to evaluate the importance of nursing, feeding and parenteral administration of fluids in the treatment of otitic sepsis it was not possible to go to the literature, for little has been written about these valuable adjuncts to treatment, which most physicians take for granted. In order to crystallize various opinions on these points a questionnaire was sent to representative otologists in various parts of the country, but, as so often happens with questionnaires, the replies were few. If, therefore, the more or less composite opinions expressed in this portion of the symposium do not meet with the approval of the hearers they will have ample opportunity to elaborate on them in the general discussion to follow.

NURSING

The opinions on the value of careful nursing vary from that of a physician who said, "It seems to me that good nursing is generally taken for granted," to the remark of Dr. Friesner, who wrote, "This enclosure was written by a Mt. Sinai nurse, to whose care, more than to my operative intervention, I owe the recovery of one of my friends." The selection of the nurse is important and should not be left to chance. In this selection one should not only be guided by the qualifications of the nurse but also by her personality in relation to that of the patient. Since a patient with otitic sepsis is usually irritable, it may be necessary to make several changes in nurses until the optimum combination is obtained.

Cooperation between the surgeon and the nurse is of prime importance and depends on both but mostly on the surgeon, who must at all times anticipate what may happen in the next twenty-four to forty-eight hours. This need not be discussed with the patient or his family, but the nurse in charge should know what complications to expect and what to watch for. The failure of patients to receive good nursing can in many instances be laid directly at the door of the surgeon. It is his business to explain to the nurse exactly what he expects her to do and that it is her duty to report to him immediately any unusual change in

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the patient's condition. It is poor cooperation between surgeon and nurse if he is not fully aware of some complication that has occurred since his last visit. The best type of nurse is one who carries out the surgeon's orders but gives the patient plenty of leeway in meeting his demands. Such a nurse can do much in maintaining the morale of the patient, who is uncomfortable, in pain, irritable and apprehensive. Without specifically discussing the condition with the patient the nurse can do much to allay apprehension by encouragement as to the ultimate outcome, the recovery of the hearing, the lack of disfiguring scars and so forth.

Certain routine duties of the nurse may, I think, be taken for granted. It is expected that she will know how and when to take the temperature and the pulse and respiratory rates and properly chart them. Even in these matters the surgeon must give her special orders if he wishes the data recorded more frequently than the usual hospital routine calls for. She is expected to keep the room at a proper temperature (about 68 F.), to keep the patient out of direct drafts, to change gowns and bed linen when they become moist or soiled and, in general, to see that the patient does not become chilled. The covering must be warm enough to prevent chilling but light enough to avoid irritating the patient by the weight of the bed clothes. Most patients with otitic sepsis are already suffering from an infection of the upper part of the respiratory tract, which must be carefully watched to prevent pneumonia and bronchopneumonia.

Maintaining the general comfort of the patient is of great importance. In many patients with otitic sepsis the sternocleidomastoid muscle has been traumatized at operation. Any movement of the head creates pain. Nurses should be instructed to keep the patient in the dorsal position with the head slightly raised to lessen tension on the muscles of the neck. A "doughnut pillow" placed on top of the regular pillow may help much to relieve this strain. The nurse should exercise great care in lifting the head or changing its position. During the first few days the patient may be more comfortable if turned on the side opposite to that operated on and if the head is supported and bolstered with small pillows. She should, on order, limit the amount of company and sedulously guard against injudicious remarks by family or friends.

The nurse should maintain meticulous asepsis in the matter of daily dressings. She should provide sterile dressings, sheets and gloves for the surgeon. If he has any special idiosyncrasies in applying dressings or putting on bandages she should acquaint herself with them to avoid any delay in the one thing which the usual patient fears the most, the daily dressing. The usual antiseptic solutions for cleansing the wound and culture tubes should always be available on the dressing tray. If

moist dressings are called for the nurse must know how to apply them without getting the gown or bed clothes wet.

The nurse in charge of a patient with otitic sepsis should be carefully instructed in the physical signs and symptoms of complications. She should watch for and recognize (*a*) drooping of one side of the face or inability to close the eye with paralysis of the facial nerve; (*b*) irritability and nuchal rigidity with a sudden rise of temperature with meningitis, (*c*) the "picket fence" temperature and chills with sinus thrombosis, (*d*) swelling below the dressings as a sign of Bezold's abscess, (*e*) Gradenigo's syndrome, (*f*) the appearance of aphasia, slow pulse and slow cerebration with abscess of the brain, (*g*) vertigo and nystagmus denoting labyrinthine invasion and (*h*) headache, for which she should have a profound respect no matter how trivial the complaint. She should not content herself with simply noting these observations on her chart but should immediately get in contact with the surgeon.

FOOD IN CASES OF OTITIC SEPSIS

Food is anything which when taken into the body serves to nourish or build up the tissues or to supply heat. Any diet should be well balanced. It should supply proteins, carbohydrates, fats, fluid and minerals. It is important to remember that the term nutrition means more than diet alone, for it also implies the transportation of assimilated food to all cells and tissues of the body. Therefore, the food offered the patient must supply all the constituents mentioned and still be offered in such form and amounts that it will be properly digested and assimilated. Most physicians are agreed that the diet should be liberal and of a high caloric content, although one of those to whom the questionnaire was sent expressed himself as not in favor of forcing convalescence as much as he had been taught while an intern.

The old adage "to force fluids and drown the fever" still seems to be good therapy, although under certain conditions even this dictum must be modified. The patient with otitic sepsis is functioning at a higher metabolic level than the healthy person. He is dehydrating himself more rapidly than normally, particularly if much nausea and vomiting are present. While nausea and other signs of acidosis are present one must content oneself with fluids, such as fruit juices, and with sugar given at frequent intervals. If this increases the nausea such nourishment must be given by other routes, such as the bowel or the vein or by hypodermoclysis. As nausea ceases the diet can be increased in bulk and in amount, but the consensus seems to be that small feedings every two hours are much better borne and assimilated than large meals at less frequent intervals. Raw fresh scraped beef and fresh meat juices are valuable for their nuclein content and to stimulate the formation of white blood cells. In the winter vitamins must be added to the diet, and if trans-

fusions have not been given the importance of blood-building and iron-containing foods must receive serious consideration.

These remarks cover the main objectives in the feeding of patients with otitic sepsis, and it should not be necessary to go into detail about the various articles of diet, but it should be remembered that nutrition in these cases actually means balanced metabolism.

PARENTERAL ADMINISTRATION OF FLUIDS

The indications for blood transfusions and the parenteral administration of dextrose and Ringer's solution are sepsis, dehydration, acidosis and malnutrition.

In every case of otitic sepsis the intake and output of fluid should be carefully charted, and when this cannot be accurately done I know of no better method of determining the need for parenteral administration of fluids than the concentration of the urine. I quote from Hartmann [1]:

> . . . regardless of the type of infection, we are likely to see, in infants and children, dehydration, fluid loss, with bicarbonate reduction and varying degrees of retention of waste products. If there . . . happens to be some kidney damage . . . these chemical changes will become pronounced, and include great reduction in chloride, N.P.N. of over 200 milligrams per 100 cc., and phosphate retention. . . .
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> In brief, if one has any kidney function at all worth speaking of, one can prevent the water changes and the electrolyte changes by the proper administration of fluids, but by fluids we do not mean simply normal saline solution or dextrose solution. . . . One may have to give . . . alkali which is generated independently of any renal activity, and which takes care of an acidosis. . . .
>
> Our own practice has been to supply in sufficient quantities such substances as sodium lactate, which can develop bicarbonate after metabolism, and Ringer's solution, which offers the body at least all the building stones necessary in the form of water, sodium, potassium, magnesium, calcium chloride, and alkali. . . .

Frank [2] stated the belief that the majority of patients with severe sepsis are best treated by intravenous injections. He stated a preference for a 5 per cent solution of dextrose in physiologic solution of sodium chloride, using 3 to 4 quarts (2.8 to 3.8 liters) daily for an adult and 2 to 3 quarts (1.9 to 2.8 liters) for a child. He stated a preference for the 5 per cent solution over the 10 per cent because, when given in large amounts, the latter spills over and is recovered from the urine. After giving the dextrose in saline solution for a time he alternates it with dextrose in triple distilled water to prevent edema due to excessive chlorides.

Acidosis can easily be diagnosed by the nausea, vomiting and the presence of acetone and diacetic acid in the urine, and in the treatment of this condition nothing is of greater value than the parenteral administration of sugar solutions.

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1. Hartmann, A. F.: Panel Discussion: Septic Thrombophlebitis of the Sigmoid Sinus; Medical Treatment, Tr. Am. Acad. Ophth. **42**:443, 1937.
2. Frank, I.: Personal communication to the author.

TRANSFUSIONS OF BLOOD IN THE CARE OF PATIENTS AFTER OPERATIONS FOR OTITIC SEPSIS

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DETROIT

The part of the symposium which the president of the American Laryngological, Rhinological and Otological Society assigned to me deals with the use of transfusions of blood in the care of the patient after operations for sepsis of otitic origin. As a procedure of biologic therapy, the transfusion of normal or of immune blood plays an important part in both the prophylaxis and the treatment of surgical sepsis. But a rational evaluation of the various technics of blood grouping and methods of blood transfusion often presents serious difficulties to the laboratory worker as well as to the clinician. Experimental evidence offers only elementary aid in clarifying the problems, since septicemia in the animal varies considerably from that in man and biologic reactions are extremely difficult to duplicate in vitro.

The growing demand for closer association of scientific research and experience in the sickroom—for "confirming practical physiologic facts from controlled observations in man" [1]—has been justified, within the last two decades, by the clarification of many heretofore unaccountable irregularities in the course of surgical sepsis. A cross section of present day literature is tangible evidence that the formerly detached schools of thought recognize in common that their ultimate purpose is the diagnosis and treatment of disease and that much of the accuracy in interpreting the clinical symptoms and the laboratory findings depends on an understanding of the physiologic principles involved.

This new outlook is of especial interest to the otolaryngologist, who must concede that the result in any given case of surgical sepsis depends not only on the biologic behavior and antigenic structure of the invading micro-organism but, to a greater extent, on the biologic behavior and defensive capacity of the innumerable cells and substances of the invaded patient's body—with the further complication that all these factors are inherently variable and hence interdependent.

Read as part of a Symposium on Care of the Patient After Operations for Sepsis of Otitic Origin at the Forty-Fifth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., Chicago, May 9, 1939.

1. Doan, C. A.: Clinical Implications of Modern Physiologic Hematology, St. Paul, The Bruce Publishing Company, 1936, p. 9.

FACTORS INFLUENCING SEPTICEMIA

With the first premonition of impending septicemia, a series of blood cultures is made, to determine, if possible, the causative organism and to estimate the degree of its pathogenicity, which means its ability to disturb the physiologic balance of the body fluids and tissues. An altered physiologic status requires an altered "clearing mechanism" [2] to digest or destroy the invaders parenterally and to neutralize their toxins. Normally, the circulating blood and the parenchymal organs evidence a remarkable capacity for removing foreign substances from the body tissues. Conversely, when the clearing mechanism is functionally impaired, invading organisms, even of low virulence, may produce septicemia. Ordinarily, micro-organisms, particularly streptococci of low virulence, may be intermittently present in the blood, as detected by cultures, when there is no clinical evidence of infection, especially in persons with chronic foci of infection in the tonsils, the sinuses, the apex of a tooth, the valves of the heart or the gallbladder. Concurrent with this bacteremia, acute mastoiditis may provide a rapid overflow of organisms, which cannot be effectively counterbalanced by the clearing mechanism, and sepsis results. Sepsis which persists after surgical drainage of the apparent focus of infection is due not so much to multiplication of bacteria in the blood as to constant invasion by organisms proliferating in foci which have not been eradicated, or which cannot be eradicated, by surgical intervention. Lillie [3] stated, "It is extremely important that the pathologic conditions causing sepsis be thoroughly understood if stages of the disease are to be recognized."

HEMOLYSIS BY BACTERIAL PRODUCTS

Both pathogenic and nonpathogenic bacteria produce hemolytic substances that are excreted into the fluids in which they grow. During severe infectious processes, marked hemolysis occurs, especially in conditions accompanied by septicemia. The hemolytic bacterial toxins most frequently encountered in otitic sepsis are those produced by Streptococcus group A, Staphylococcus aureus and Pneumococcus type III. Wells [4] considered

> it . . . probable that the *bacterial "hemolysins" are all merely toxins with a particular affinity for red cells* . . . against some of these hematoxins antitoxic

2. Kolmer, J. A.: Etiology, Prophylaxis and Treatment of Surgical Septicemia: A Discussion of the Principles Involved, Arch. Otolaryng. **26**:59 (July) 1937.

3. Lillie, H. I., in discussion on Kopetzky, S. J.: Panel Discussion: Septic Thrombophlebitis of the Sigmoid Sinus; *A*. The Pathology; *B*. The Bacteriology; *C*. Immunological Treatment, Tr. Am. Acad. Ophth. **42**:432, 1937.

4. Wells, H. G.: Chemical Pathology, ed. 5, Philadelphia, W. B. Saunders Company, 1925, pp. 248.

sera are obtainable, although there is . . . some question as to how much of the antagonistic effect depends on true antitoxins and how much upon the cholesterol in the serum.

Streptolysin and staphylolysin unite directly with the receptors of the red cells without the intervention of an intermediary body. Zinsser [5] reported that the

action of many bacterial poisons is selective. Most of these poisons excite inflammatory reactions if concentrated in any part of the body, but in addition to this, there is specific distribution which indicates that the poison goes into selective relationship with certain tissues and cells. This fact is illustrated by the bacterial hemotoxins which specifically injure the red blood cells of the infected individual and by such substances as the leukocidin produced by the Staphylococcus aureus, a poison which injures the white blood cells.

Wells [4] stated:

with streptococci, Lyall found that the hemolysin titer did not afford a criterion of virulence. No immunity to streptococci is produced in animals immunized with streptococcic hemolysin. Pneumococci produce an intracellular hemolytic toxin which is very labile and antigenic. . . . Kämmerer found that of a large series of organisms, only pneumococcus and *Strep. hemolyticus* produce methemoglobin.

Stadie [6] found that in pneumonia enough methemoglobin may be formed to cause a decrease in the oxygen capacity of the blood, the essential cause of cyanosis in pneumonia.

The first products of the splitting of the hemoglobin are the complex protein globin (which constitutes 94 per cent of hemoglobin), and hematin, the iron-containing compound. Some of the products of disintegration of hemoglobin seem to inhibit or destroy gram-positive bacteria. Many nonspecific hemolytic substances, such as acids and bases, also may be formed by bacteria as products of their metabolism; staphylococci may produce hemolytic fatty acids by splitting fats. Wells [7] stated, "The bacterial products may also modify coagulation, and L. Loeb has found that . . . *Staphylococcus aureus* [is] . . . much more powerful in causing coagulation than any other . . . [organisms] tested." Studying converse properties, Tunnicliff [8] reported, "With the exception of some strains of Staphylococcus aureus, fibrinolysis appears to be peculiar to the hemolytic streptococcus." She found that this action appears to be associated with the virulent smooth

5. Zinsser, H.: Resistance to Infectious Diseases, ed 4, New York, The Macmillan Company, 1931, p. 41.

6. Stadie, W. C.: Studies of the Blood Changes in Pneumococcic Infections, J. Exper. Med. **33**:627, 1921.

7. Wells,[4] pp. 353-355.

8. Tunnicliff, R.: Effect of Dissociation of Streptococci on Their Fibrinolytic and Anticlotting Activity, J. Infect. Dis. **58**:92, 1936.

type. The power is lost as the organism becomes rough. Whipple [9] maintained that coagulability decreases in sepsis because of the presence of an excess of antithrombin and that the antithrombin factor is often excessive in hemorrhagic conditions, especially with hepatic impairment. Antithrombin is often increased in diseases of the blood-forming organs, e. g., leukemia, possibly as a reaction to the products of disintegration of corpuscles. Wells,[7] however, stated:

> The fluidity of the blood in septicemia is probably dependent upon the appearance of the coagulation-inhibiting phase that follows the action of the products of cell destruction, including among them proteoses.

ANEMIA DUE TO HEMOLYTIC AGENCIES

Secondary anemia occurring in the course of otitic sepsis may be explained largely by the hemolytic property of the invading bacterial products; hence, it presents quite different features from those of other secondary anemias. The red corpuscles almost solely are attacked, and the products of their disintegration are present in the plasma. As a result, the plasma or serum may contain free hemoglobin, and if this is in large amounts it may escape into the urine. The products set free by disintegration of the hemoglobin are present not only in the blood but also in the parenchymal organs particularly the liver and the spleen, which accumulate iron. Fatty changes occur in all these organs, with changes in metabolism which are similar to those observed in other forms of anemia. With increased catabolism of protein, the plasma chlorides are increased,[10] the alkaline reserve decreased and the protective power of the serum against hemolysis by saponin, fatty acids, oleates and other products of suboxidation reduced.[11] Suboxidation is manifested by the presence of acetone, oxybutyric acid and diacetic acid in the urine.

PHYSIOLOGIC EQUILIBRIUMS OF THE BLOOD

Doan [12] stated:

> The individual types of cells in blood and tissues may . . . be understood and their activities [effectively] influenced or controlled [only] as we . . . acquire . . . specific information concerning their fundamental physiologic relationships to each other and to the other cells and functions of the body. Bone

9. Whipple, G. H.: Hemorrhagic Disease—Septicemia, Melana Neonatorum and Hepatic Cirrhosis, Arch. Int. Med. **9**:365 (March) 1912; II. Hemorrhagic Disease: Antithrombin and Prothrombin Factors, ibid. **12**:637 (Dec.) 1913.

10. Steinfield, E.: The Plasma Chlorides in Anemia: An Experimental Study, Arch. Int. Med. **23**:511 (April) 1919.

11. Clark, H. M., and Evans, F. A.: Studies with Lecithin and Cholesterin in Relation to the Antihemolytic Property of Human Serum, Bull. Johns Hopkins Hosp. **32**:113, 1921.

12. Doan,[1] p. 15.

marrow, lymphatic tissue, spleen, liver, kidneys, gastro-intestinal tract, and some of the endocrine glands (pituitary, thyroid, adrenal) are now known to be intimately associated with [the] blood cell equilibria.

Disturbance of normal balance in one aggregate of cells cannot be limited to that aggregate.

As defined by Wells,[13]

> The function of the blood [is] . . . to maintain an equilibrium in the temperature, chemical composition, and osmotic pressure between all parts of the body. . . . To the extent that every tissue is continually giving off something to the blood, we may consider that every organ is a factor in its formation. . . . There are probably but few chemical substances occurring in the tissue-cells that do not also occur in greater or less amount in the blood. In addition . . . there are . . . the substances characteristic of the blood itself, besides a host of substances of unknown nature, apparently manufactured in response to the stimulation of substances entering the body from outside.

Hence the circulating cellular elements of the blood constitute an essential and powerful increment in the defensive forces of the body.

ORIGIN, MATURATION AND DISTRIBUTION OF THE VASCULAR CELLS

According to Doan [14] hematologists agree "that all [the cells of the blood] take their first beginning from the mesenchymal cells of the mesodermal layer in the embryo." He stated, "The bone marrow throughout extrauterine life is the natural source of erythrocytes, thrombocytes and the three kinds of granulocytes." The neutrophilic granulocyte plays a major role in the mechanism of defense against infection and toxemia, the defensive value depending on its maturity and the integrity of its bactericidal ferments. The parent cell, the myeloblast, consists at first of a large nucleus surrounded by a small amount of clear cytoplasm. As the cell grows, the nucleus becomes relatively smaller; the cytoplasm, more abundant; the nucleoli disappear; the granules increase in number and are smaller; the nucleus is indented, and the cell shows motility for the first time. The nucleus separates into two or more lobes, united by a thin thread or filament. The older the cell is on leaving the marrow, the more lobes it shows (although the presence of more than five lobes is uncommon). The oxidase staining is now intense, indicating the high ferment content. Mitotic activity [15] always occurs in an area of rich blood supply, between widely dilated venous sinusoids, in contradistinction to erythropoiesis, which occurs intravascularly in relatively anoxemic areas in the marrow.

13. Wells,[4] pp. 323-326.

14. Doan, C. A.: Current Views on the Origin and Maturation of the Cells of the Blood, J. Lab. & Clin. Med. **17**:887, 1932.

15. Sabin, F. R., and Miller, F. R.: Normal Bone Marrow, in Downey, H.: Handbook of Hematology, New York, Paul B. Hoeber, Inc., 1937, pp. 1791-1821.

The phagocytic macrophages (clasmatocytes), while always present in the marrow, are quite as regularly and normally found in the spleen, liver and diffuse connective tissues. They are characterized by their size and ability to arise in situ throughout the body as the need presents. Lymphocytes and monocytes, the remaining elements which utilize the vascular bed as an avenue of distribution, develop in lymph nodes, spleen and the more diffuse lymphoid and connective tissues of the body.

HEMOPOIETIC REACTION TO INFECTION

The leukopoietic response to an invading noxa of infection and toxemia is a biologic response, in that the myeloid elements react initially to the irritating forces by an increase in concentration, to be followed by greater activity of the monocytes and lastly of the lymphocytic elements. This response is reflected in the reactions of the leukopoietic centers as well as in the erythropoietic tissue.

While it is admitted that the typical response of the leukopoietic system to infection is leukocytosis and neutrophilia, it must be remembered that an atypical response may result from a prior myeloid depression or an abnormal lymphocyte-monocyte ratio or that anemia may have preceded the development of otitic sepsis. The absence of hyperleukocytosis and neutrophilia concurrently with dynamic toxemia warrants neither an assumption that there is an inadequate immunologic response nor its converse.

In an evaluation of the blood pattern in otitic sepsis it is mandatory that the first consideration be given to the concentration of all the blood elements: Are they present in normal quantity, or are they decreased or increased? The second step is to compare the quantitative relation of the formed elements: Are the erythrocytes, hemoglobin and thrombocytes uniformly reduced, or is one of these elements appreciably depleted, thereby creating a marked disequilibrium? An identical appraisal is made of the white blood cell pattern, after which an intensive search is pursued for the detection of morphologic changes in the cellular components (Sharp [16]).

SHIFTS TO THE LEFT: "TOXIC" GRANULATIONS: WEAKENED DEFENSES

Whether or not the leukocytes are present in abnormal concentration, one of the primary reactions of the myeloid centers to infection is a tendency for the mature neutrophils to be replaced by those of a primitive status. Instead of polylobular neutrophils dominating the

16. Sharp, E. A.: Hematopoietic Reaction to Infection, unpublished data; personal interview with the author.

myeloid series in the peripheral blood pattern, metamyelocytes and myelocytes appear. This is designated as a "left" myeloid shift, and it may occur with neutrophilia, with neutropenia or when the total concentration of myeloid elements does not exceed the normal range, 58 to 66 per cent of the total number of white cells.

When toxemia is more than transitory, basophils and eosinophils are decreased to absence. The lymphocytes and monocytes, with exceptions, are inclined to decrease during the dynamic phase of infection, particularly when neutrophilia is appreciable. Having determined these points, the morphologic characteristics of the individual cell series are studied: Are "toxic" granulations present in the cytoplasm? Is the nucleus in each cell series normal in size and structure?[16] Haden[17] interpreted the qualitative changes in neutrophilic leukocytes as follows:

> . . . A neutrophilic leukocytosis with mature normal cells indicates the presence of an infection or toxemia to which the marrow is fully able to respond.
>
> . . . A neutrophilic leukocytosis with less mature cells or a significant increase in immature cells without an increase in the total count indicates either:
>
> a. Some difficulty in response of the marrow to an infection or toxemia.
>
> b. An overwhelming infection or toxemia requiring the marrow to deliver many granulocytes. . . .
>
> . . . A leukopenia with immature cells indicates a depression of the marrow preventing the normal reaction to infection or toxemia.
>
> . . . The presence of "toxic" granulation with or without leukocytosis and regardless of the maturity of the cells, indicates, [the presence and the severity of] an infection or toxemia which is injuring the marrow.

The reappearance of the basophils and eosinophils presages convalescence.

CLINICAL PICTURE IN OTITIC SEPSIS

The clinical symptoms are manifested in varying degrees, depending on the extent and intensity of the infection and the causative pathogen. The common streptococcic or staphylococcic infection is, as a rule, first local, and the toxins alone pass into the blood. Symptoms of the invasion of the blood and the lymphatics by the bacteria usually set in within twenty-four hours and rarely later than the third or fourth day after the operation. There is a chill or chilliness with moderate fever at first, which may gradually or suddenly increase and is marked by daily remissions and even intermissions, with chills and drenching sweats. Sometimes symptoms suggesting involvement of the cerebrum dominate the picture. In other cases, gastrointestinal disturbances, such as nausea and vomiting, predominate. As the disease progresses the pulse becomes small, rapid and compressible. There may be periods of

17. Haden, R. L.: The Interpretation of Qualitative Changes in Neutrophilic Leukocytes, Cleveland Clin. Quart. **5**:184, 1938.

delirium or marked mental prostration and apathy. There may be pallor or a yellowish tint of the face, lips and finger tips. The mouth is parched; the tongue is red at the margin, and the dorsum is dry and dark. A mild or severe rash or even capillary hemorrhages may appear over the surface of the body.

INDICATIONS FOR TRANSFUSIONS OF BLOOD

In otitic sepsis toxic substances gradually saturate the parenchymal organs; the combustive processes are incomplete, resulting in subnormal elimination of waste products and the toxins of bacterial decomposition. The cellular elements of the body are in drastic need of oxygen, nutrition and water, and since physiologic equilibrium depends on an adequate supply of oxygen, nutrition and fluids, which can be furnished only by the blood, it is reasonable to assume that whole human blood is theoretically and practically the ideal fluid for transfusion. The amount administered, the interval between transfusions, the condition of the patient, the qualitative changes in the blood and especially a reduced concentration of hemoglobin should all be correlated with the indications for transfusion.

The purpose of transfusions of blood is to counteract the following conditions:

1. Hemorrhage, traumatic, secondary to the local disease or secondary to operation.

2. Shock, surgical [18] or hemolytic.[19]

3. Anemia complicating the local disease.

4. A progressive, falling concentration of hemoglobin.

5. Toxemia complicating mastoiditis or thrombosis of a sinus or jugular vein.

6. Debilitation, with or without secondary anemia.

7. A loss of plasma proteins associated with acute anhydremia, especially in infancy and childhood.[20]

8. Acute hemolytic anemia secondary to the use of sulfanilamide, for which blood transfusions should be given until the blood level is restored (Shurly [21]).

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18. McFee, W. F., and Baldridge, R. R.: Post-Operative Shock and Shock-Like Conditions: Treatment by Infusion in Large Volume, Ann. Surg. **91**:329, 1930.

19. Krampf, F.: Hemolytic Shock Following Blood Transfusion, Wien. klin. Wchnschr. **51**:407, 1938.

20. Hartmann, A. F.: Panel Discussion: Septic Thrombophlebitis of the Sigmoid Sinus; Medical Treatment, Tr. Am. Acad. Ophth. **42**:443, 1937.

21. Shurly, B. R.: Personal interview with the author.

THERAPEUTIC VALUE OF TRANSFUSIONS OF BLOOD

In general, the therapeutic value of transfusions of blood depends on the following actions:

1. They supply serum and plasma proteins which aid in restoring colloid pressure, blood volume and water balance within the vessels, thus diluting toxic blood.

2. They provide immediately an available supply of oxygen carriers.

3. They furnish a quantity of fresh leukocytes for the elaboration of bactericidal enzymes, opsonins which render organisms susceptible to phagocytosis, agglutinins which flocculate organisms, thus favoring phagocytosis, and bactericidal plasma or serum to dissociate [22] or degrade bacteria to a form which can be more readily phagocyted; also they furnish such nonspecific bactericidal agents as the leukins and the plakins.

4. They stimulate the hemopoietic centers, thus aiding the formation of white and red cells.

5. They support hepatic function, which is essential to the blood and the hemopoietic efficiency.

MINERAL AND CARBOHYDRATE ADJUVANTS TO BLOOD THERAPY

1. Since "water regulation of the body . . . is intimately linked with the metabolism of sodium and potassium and [with] the . . . function of the kidney," [23] and since in severe septic conditions there is, as a rule, considerable dehydration, due to forced loss of extracellular fluids and bases, physiologic solution of sodium chloride or Ringer's solution may be given intravenously to maintain fluid balance. Unless urgently needed, no more than 500 cc. is given in one transfusion. Hypertonic solutions are employed only when specifically indicated. Should a blood transfusion be required during the period of intravenous therapy, it may be given without withdrawing the needle, unnecessary venipuncture thus being avoided. When a secondary operation is necessary for severe or prolonged otitic sepsis, it is advisable to give a transfusion of 150 cc. to 250 cc. of whole blood for adults and 100 cc. to 150 cc. for children preoperatively. A similar amount should be administered immediately after the operation and the procedure repeated every second day as indicated. Loss of blood in such patients will produce symptoms out of all proportion to the amount of blood lost at the time of operation. Loss of other body fluids at this time may further complicate the picture. Shock may thus be prevented, and what appeared

22. Mellon, R. D.: A New Approach to the Therapeusis of Hemolytic Streptococcus Infection, Proc. Soc. Exper. Biol. & Med. **34**:474, 1936.

23. McCance, R. A.: Medical Problems in Mineral Metabolism (Goulstonian Lectures), Lancet **1**:643, 704, 765 and 823, 1936.

to be a poor surgical risk may now become a good one. Blood volume is bound up with hemorrhage and shock. Lewisohn [24] asserted that if both transfusion and surgical procedure are necessary, the transfusion should be given first.

2. The blood sugar level should be maintained by the addition of dextrose to the sodium chloride solutions as indicated. Fats and carbohydrates are the first drawn on and later the proteins, in an effort to provide water for the maintenance of the normal fluid content. When conditions arise which tend to lower or raise the blood volume, either the hydrostatic or the osmotic pressure or both are altered, and it is through such changes that the blood fluid is automatically restored to its previous level (Best and Taylor).[25]

According to Hartmann [20] the course of otitic sepsis, especially in infancy and childhood, may be complicated by (1) "severe disturbances of water balance and acid base balance," (2) depletion of carbohydrate stores permitting ketosis and even severe hypoglycemia, which interferes "with efficient circulation of the blood," (3) lowering of plasma volume through "loss of water . . . or loss of protein from the plasma" and (4) "ineffectual circulation because of loss of red cells from anemia." Hartmann stated also:

> One may find acute anhydremia with circulatory failure in the presence even of edema, . . . because the level of plasma protein is not sufficient to bring water back from the tissue into circulation, and to correct a change like this will require the transfusion of whole blood, with sufficient plasma, or of plasma itself repeated often enough, or the transfusion of some substitute for plasma, such as [a 6 per cent solution of] acacia.

SELECTION OF A COMPATIBLE DONOR

The routine procedure of a preliminary blood grouping followed by a direct matching of the donor's cells and the recipient's serum has been firmly established. With high titer serums, this matching detects the presence in the recipient's serum of atypical agglutinins which may be active against the prospective donor's cells. When the same patient is to be given more than one transfusion, even if the same donor is to be used again, the cross agglutination should be repeated before each transfusion. In addition to compatibility, the quality of the donor's blood should be appraised for hemoglobin content and dyscrasias of blood and bone marrow. Collier [26] and others have advocated a careful medical history, physical examination and laboratory studies to exclude

24. Lewisohn, R., cited by Wiener,[47] p. 51.

25. Best, C. H., and Taylor, N. B.: The Physiological Basis of Medical Practice, Baltimore, William Wood & Company, 1937, pp. 22-31.

26. Collier, W. D.: The Uses and Abuses of Transfusion, Ohio State M. J. **34**:1111, 1938.

prospective donors with transmissible diseases, "such as syphilis, malaria, virus, bacterial and allergic diseases." It is now known that the serologic test for syphilis is not sufficient, since "the chancre may be present and the blood full of spirochetes although the blood serology is not yet positive." It has been found advisable to exclude the "emotionally unstable donor" and donors who have partaken of food or alcoholic beverages within five hours before the taking of blood.

CONTRAINDICATIONS FOR BLOOD TRANSFUSIONS

With the altered conceptions of contraindications for blood transfusions which have evolved with the newer collaboration of research worker and clinician within the last two decades, the otolaryngologist finds increasing need to consult the hematologist, the internist, the pathologist and the pediatrician in cases of precarious otitic infection. Obviously the otolaryngologist does not attempt the independent supervision of any intravenous injection when there is pulmonary edema or serious myocardial degeneration. In such cases transfusions, if given, must be administered at an extremely slow rate and only by trained transfusionists.

The frequent injury of the heart muscle by bacterial poisons or the irregular parenchymatous changes in various organs may be determined, according to Zinsser,[27] by functional activity and increased metabolism. In many infectious conditions the bacteria themselves pass through the kidney into the urine, and "renal injury may result from the actual presence of the bacteria in the kidney," as well as from the lesions produced by bacterial poisons in the course of excretion. Severe otitic infections requiring transfusions may be seriously complicated by acute nephritis. The older opinion that blood transfusion should not be used in cases of nephritis has been refuted by many authors. Cooksey [28] asserted:

> Certainly in the early days of acute hemorrhagic nephritis, blood is of very real assistance. . . . in any kind of nephrotic edema, even that of chronic Bright's disease, blood may sometimes be given to raise the serum protein levels with value. However, always caution must be used in transfusing cases of nephritis, for if a severe reaction should occur, with further damage to the renal tubules, a grave condition may result.

Others withhold transfusions from the asthenic and the aged and consider certain dyscrasias of the blood, as leukemia, a contraindication to transfusion except as a temporary stimulant.

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27. Zinsser,[5] p. 46.

28. Cooksey, W. B.: Recent Advances in Blood Transfusion, J. Michigan M. Soc. **38**:409, 1939.

METHODS OF TRANSFUSION OF BLOOD

The following forms of blood for transfusion have been recommended and used in the treatment of sepsis of otitic origin and in other conditions requiring blood therapy:

1. Whole unmodified blood (direct transfusion).
2. Citrated or modified blood (indirect transfusion).
3. Specifically immunized blood (impractical except for prolonged sepsis).
4. Nonspecifically immunized blood.
5. Immunized blood and blood serum from convalescent donors.

A recent survey of blood transfusion in America, compiled by Levine and Katzin [29] from data received from three hundred and fifty approved hospitals, evidenced a general increase in the number of transfusions being given and a decided preference for the citrate method. Several reasons are given for the popularity of this method, chief of which is the expressed belief that no special precautions are required. The authors stated:

> For patients suffering from infections of the blood stream, the citrate method is used, apparently because it affords absolute protection to the donor. On the other hand, some of the advocates of the direct method believe that the advantages of the citrate method are more apparent than real and maintain that whole blood is preferable for patients with septicemia.

Bipartisan authors have found no essential difference in therapeutic value between modified whole blood and unmodified whole blood when transfused. To ameliorate and control the bacteremia or septicemia complicating thrombosis of the lateral sinus, Coates, Ersner and Persky [30] advocated the use of blood transfusions and affirmed: "It is immaterial whether one uses the whole or the citrated blood." Small transfusions (in adults, 150 to 250 cc.; in children and infants, "1 to 1½ cc. of whole blood per pound of body weight") may be given preoperatively or postoperatively "and should be repeated at frequent intervals, particularly where the infection is very severe." Indications for postoperative transfusions are a "persistent septic type of temperature, . . . increasing anemia, or a marked loss of resistance."

Lillie [31] advised, for a "very sick and washed out" patient,[31a] delaying operation "in order to improve the general condition by using fluids

29. Levine, P., and Katzin, E. M.: A Survey of Blood Transfusion in America, J. A. M. A. **110**:1243 (April 16) 1938.

30. Coates, G. M.; Ersner, M. S., and Persky, A. H.: Lateral Sinus Thrombosis, with Review of the Literature, Ann. Otol., Rhin. & Laryng. **43**:419, 1934.

31. Lillie, H. I.: The Surgery for Sepsis of Otitic Origin, in Nelson New Loose-Leaf Surgery of the Ear, New York, Thomas Nelson & Sons, 1938, pp. 261-287.

31a. Lillie, H. I., in discussion on Hartmann,[20] p. 455.

and blood transfusions." In the postoperative treatment of otitic sepsis he stressed the importance of carefully determining the concentration of hemoglobin, as a definite decrease in this element of the blood indicates a serious disturbance of physiologic balance. "One cannot set up a definite standard for the concentration of hemoglobin at which transfusion of blood is indicated, but the falling concentration of hemoglobin and the general appearance of the patient are rather good indices."

In the postoperative treatment of thrombosis of the lateral sinus, Kopetzky [32] concurred with others in the use of blood transfusions for complicating anemia but expressed disagreement with those who prescribe blood transfusions to counteract a septic temperature. He stated the belief that a persisting septic temperature curve indicates that the focus of infection has not been eradicated and that repeated transfusions are a hindrance to speedy recovery. On the other hand, Pemberton [33] found that transfusions given shortly after, and in some instances before, the operation offer a reasonable means of combating acute toxic and septic conditions. In comparative view, Friesner [34] asserted that blood transfusions contribute only indirectly to the cure of otitic sepsis and "contribute no more than food or any other supportive measure does." Fenton [35] advocated, for children, supportive transfusions of 150 cc. of "whole citrated blood" preceding the operation and transfusions repeated every third or fourth day postoperatively. He found "no special reason to crowd transfusions . . . unless the hemoglobin index has fallen below 60." After surgical treatment of the mastoid, the sigmoid sinus and the jugular vein, Furstenberg and Maxwell [36] gave the patient supporting treatment including small transfusions of "whole blood" at two or three day intervals, from different donors. Reduced iron in doses of 0.5 Gm. was administered orally three times a day, and fluids were forced to 4 or 5 liters a day for adults. Patients unable to take adequate fluids by mouth were given a continuous intravenous injection of 5 per cent dextrose. Dissipated body chlorides were replaced by physiologic solution of sodium chloride administered intravenously.

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32. Kopetzky, S. J.: Acute and Chronic Otitis Media and Sinus Thrombosis, Arch. Otolaryng. **10**:302 (Sept.) 1929.

33. Pemberton, J. de J.: Blood Transfusions, Surg., Gynec. & Obst. **28**:262, 1919.

34. Friesner, I.: Infections of the Middle Ear: Acute Systemic Infections from the Ear, Arch. Otolaryng. **14**:257 (Sept.) 1931.

35. Fenton, R. A.: Some Observations on the Management of Infections of the Blood Stream from Mastoiditis, Arch. Otolaryng. **25**:618 (June) 1937.

36. Maxwell, J. H.: Thrombosis of the Sigmoid Sinus: Clinical Analysis, Arch. Otolaryng. **25**:184 (Feb.) 1937; Management of the Septic Patient with Otitis Media, J. A. M. A. **110**:1536 (May 7) 1938.

IMMUNE BLOOD AND SERUM THERAPY

In view of the increasing use of specific therapeutic agents in the supplementary treatment of surgical septicemia, Wood [37] emphasized

> the importance of determining the exact nature of every invading organism. This is a prime essential for specific therapy, whether biologic or chemical. With such information recorded, the person who has recovered is a possible donor of blood for another person suffering from the identical infection. The value of the immune substances in the new blood depends on its specificity. [Hence,] . . . not only . . . the type . . . but . . . the strain of the [invading] bacteria . . . should be determined exactly and recorded.[37]

The suggestion recently made by Gill [38] for a system of universal specific therapy would utilize the antibodies of patients who have survived similar infections. He reported:

> Transfusion of immunized whole blood should be given for its prophylactic value to persons with lowered resistance, as indicated by the opsonic index and the blood picture. . . . In cases of infection of the blood stream and the fixed tissues, transfusion of immunized whole blood has a definite curative value and should be administered every other day until the well-being of the patient justifies discontinuance.

In some instances the direct method of transfusion was used; antistreptococcus serum, given intramuscularly, was utilized freely, and a solution of dextrose and sodium chloride was used intravenously.

In the postoperative treatment of otitic septicemia, Lynch [39] selected as donors those who had had scarlet fever, erysipelas or septic sore throat, and whose serum showed the highest opsonic index, providing the blood grouping and cross matchings indicated suitability. Small transfusions, from 150 to 200 cc., were administered every eight hours for twenty-four hours and "then 300 cc. daily or every other day as the patient's condition warrants." In addition, he gave injections of antitoxin in cases of severe toxicity. He reported gratifying results in a series of 10 cases. Reverchon [40] also reported favorable results in regulating septic temperature and in improving the general condition in patients treated similarly.

Crocker, Valentine and Brody [41] presented "a preliminary report on fifty-two cases of septicemia and acute infections treated with hemog-

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37. Wood, E. L., in discussion on Gill.[38]

38. Gill, E. G.: Infection of the Blood Stream, Arch. Otolaryng. **27**:67 (Jan.) 1938.

39. Lynch, M. G.: Immunotransfusion in Otology, New Orleans M. & S. J. **91**:89, 1938.

40. Reverchon: L'immunotransfusion dans le traitement de la septicémie d'origine auriculaire, Ann. d'oto-laryng., 1932, p. 1133.

41. Crocker, W. J.; Valentine, E. H., and Brody, W.: Hemography-Controlled Nonspecific Immunotransfusions in the Treatment of Septicemia and Other Acute Infections, J. Lab. & Clin. Med. **20**:482, 1935.

raphy-controlled nonspecific immunotransfusions." The object of their paper was stated as follows:

1. To show that cases, in which this treatment is indicated, can be diagnosed and controlled best when shiftograms are used in conjunction with clinical findings and other laboratory data.

2. To show that nonspecific immunotransfusions may be used successfully in the treatment of acute infections which are of such rapid progress and great severity that there is not time to prepare a specifically immunized donor, vaccine or other biologic substance.

3. To point out the fact that unless there is adequate natural or surgically established drainage of the foci of infection, the beneficial effect of the nonspecific immunotransfusions is only transitory. . . .

Daily hemograms were done and charted and each case was closely controlled by hemograms before and after transfusion. In this manner a change of the patient's condition for better or worse was often detected several hours before it had appeared clinically. A second donor could then be prepared for transfusion while the patient's condition was still fair.

They expressed a preference for the direct method of transfusion and stated:

In severe rapidly progressive infections small transfusions (100 to 250 cc.), at intervals of six to twelve hours are of value. . . . Transfusions are repeated daily until the shift is forced right to a Schilling index of less than one. . . . Of 52 patients thus treated 25 recovered and 27 died. Five reasons appear for failure of recovery: treatment too long delayed, overwhelming infection, insufficient number of donors available, impossibility of adequate drainage and extensive pathology of incurable character.

J. E. Gordon [42] advocated the use of whole unmodified blood and blood serum from persons recently convalescent from scarlet fever to combat scarlet fever and its complications, including otitic sepsis. Blood was not used earlier than the fifteenth day of the donor's illness. Usually one transfusion produced the desired effects; in other cases two or three transfusions were required as indicated. Harrell [43] more recently reported favorable results after injection of 30 cc. of convalescent human serum into the fascia lata in cases of toxic and septic mastoiditis due to the hemolytic streptococcus, both contagious and noncontagious. His findings indicate that "convalescent serum contains anti-bodies which may not be bacteriologically specific, but therapeutically specific against streptococcic infection, which may clinically be a different disease from that from which the donor has recovered."

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42. Gordon, J. E.: Immunotransfusion in Scarlet Fever, J. A. M. A. **100**: 102 (Jan. 14), 1933.

43. Harrell, V.: Hemolytic Streptococcic Mastoiditis in Contagion and Noncontagion, Ann. Otol., Rhin. & Laryng. **46**:194, 1937.

W. H. Gordon [44] and others who used specifically immunized blood in the treatment of typhoid fever and scarlet fever found a high incidence of recovery.

Tillett and Stock [45] "demonstrated that sera from patients acutely ill with different [streptococcic] infections were capable of destroying hemolytic streptococci of the *beta* type" but that the bactericidal action disappeared "when the stimulus of active disease was no longer present. . . . It was noted, in addition, that the streptococcidal potency was greatest in samples of sera derived from patients with the most severe types of illness." Tillett [46] stated:

> The bactericidal property of the human serum which Stock and I have studied differs from an antibacterial immune response in the ordinary sense of the word. The action which we observed is present only during the acute phase of illness and disappears during convalescence, when specific antibodies usually make their appearance. Consequently, if the property which we observed has any value, it cannot be transferred from convalescent patients, since it is not there at that time.
>
> The question of immunity to the hemolytic streptococci is unusually complicated.
>
> For example, strains of hemolytic streptococci appear to have a type specificity which is in some respects comparable to the type specificity of pneumococci but which also differs from it in some respects. Twenty-eight different types of hemolytic streptococcus have been described by Griffiths. It is apparent from this fact alone that a so-called immune donor would have to have immunity corresponding to the type of organism infecting the prospective recipient.

ADVERSE EFFECTS OF TRANSFUSIONS OF BLOOD

Although much has been learned concerning the cause of adverse post-transfusion reactions, there are some which cannot always be avoided even in the absence of contraindications. It is generally conceded that the origin of most of these reactions is referable to (1) the use of specifically incompatible blood or (2) the presence in the donor's blood serum of nonspecific protein components and other substances which when transfused produce reactions suggesting allergic phenomena. In addition to these, Wiener [47] has suggested as possible causes of post-transfusion reactions: (3) the use of citrate for transfusions, (4) incipient coagulative changes in the transfused blood, (5) minor irregularities in blood grouping not detected by routine tests, e. g. anomalous

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44. Gordon, W. H.: The Use of Immunotransfusions in the Treatment of Scarlet Fever and Typhoid Fever, unpublished data.

45. Tillett, W. S., and Stock, C. C.: Bactericidal Action of Human Serum on the Hemolytic Streptococcus, J. Exper. Med. **66**:617, 1937.

46. Tillett, W. S.: The Question of Immunity to Hemolytic Streptococci, personal communication to the author.

47. Wiener, A. S.: Blood Groups and Blood Transfusions, Springfield, Ill., Charles C. Thomas, Publisher, 1935, pp. 56-61, 26-35 and 44.

agglutinins not conforming to the scheme of the four blood groups, (6) foreign matter derived from the apparatus, (7) the introduction of too large an amount of blood, which leads to overburdening of the circulation and even to pulmonary and cerebral edema, (8) the development of isoantibodies in the plasma of persons who had previously received injections of compatible blood and (9) indiscriminate use of the universal donor, group 0, whose agglutinins are of high titer. Another cause may be (10) the factor suggested by Polayes and Lederer:[48]

> Weak agglutinogens or agglutinins in the recipient's blood especially in the case of infants may also lead to false group determinations. Failure to recognize this fact has resulted in reactions after repeated transfusion in children in whom the agglutinins developed or became active after the first transfusion.

1. In their survey of hospitals, Levine and Katzin[29] found:

> The largest group of [post-transfusion] accidents was attributable to the injection of incompatible blood. . . . in the vast majority of instances the grouping serums employed are not potent and/or the persons performing the tests are not sufficiently trained.

In discussing post-transfusion reactions, Wiener[48a] asserted that it is "practically impossible to miss positive reactions if the testing sera are of high titer" but that stock serum may be of high titer when stored in vials yet the titer may fall before the serum is used. Since serum deteriorates, even when kept under sterile conditions, the contents of each vial must be tested against known cell suspensions before use. However, Gonzales, Vance and Helpern[49] stated:

> Incompatible bloods have been employed for transfusions either because the iso-agglutination tests were not performed with sufficient care, or because an aberrant hemolysin in the serum of the donor could not be demonstrated in the preliminary tests. . . .
>
> The effect of transfusing a specifically incompatible blood into the circulation of a recipient is to cause an agglutination and hemolysis of the red blood cells followed by the blocking of the renal tubules with blood pigment, [leukocytes and other desquamated cells].

Bordley[50] concluded from personal study

> of the complete records of three cases and from the available information concerning fourteen others . . . that the characteristic delayed reaction following transfusion

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48. Polayes, S. H., and Lederer, M.: Reactions to Blood Transfusions, J. Lab. & Clin. Med. **17**:1029, 1932.

48a. Wiener,[47] p. 26.

49. Gonzales, T. A.; Vance, M., and Helpern, M.: Legal Medicine and Toxicology, New York, D. Appleton-Century Company, Inc., 1937, pp. 422-423.

50. Bordley, J., III: Reactions Following Transfusion of Blood, with Urinary Suppression and Uremia, Arch. Int. Med. **47**:288 (Feb.) 1931.

. . . [in these cases was] dependent on the injection of incompatible blood [; and] . . . that most of the signs and symptoms . . . [of] reaction . . . develop from the severe functional damage that the injected blood inflicts on the kidneys.

However, definite incompatibility was established in only 6 of the 17 cases reported. From their observations of 2,500 transfusions, Polayes and Lederer [48] asserted:

To ascribe all reactions of this type to incompatible blood would . . . not account for [certain fatal cases in our experience in which the transfused blood was found by previous cross agglutination tests to be compatible with the recipient's]. . . .

The changes noted in the tubules of the kidney, which are so similar to those of the so-called tubular nephritis, described . . . as occurring in pyloric obstruction with vomiting, has lent support to the theory that the post-transfusion renal insufficiency follows a loss of chlorides incident to vomiting and its consequential nitrogen retention.

DeGowin's [51] analysis of 3,500 blood transfusions attributed 7 deaths directly to the transfusions: 5 to renal insufficiency and 2 to pulmonary edema. He reported also 3 recoveries in cases in which the patient had developed hemolytic reactions without renal insufficiency. He concurred with other authors that the present laboratory tests as routinely applied are inadequate in certain instances to detect incompatibilities of bloods, which, after transfusion, become manifest clinically. "In view of our ignorance of the mechanism of the renal damage it would seem that the only safe procedure at present is to alkalinize the urine prior to transfusion."

Gonzales, Vance and Helpern [49] further noted:

The symptoms [of renal lesions due to the transfusion of a specifically incompatible blood are] . . . tingling pains over the entire body, fulness of the head, oppressive precordial pain, and severe pain in the lumbar region. The face is markedly cyanotic, the respirations are labored, the pulse is slow, . . . 20 to 30 times a minute . . . a chill, high fever (103° to 107° F.), . . . jaundice, delirium and shock supervene. Hemoglobinuria is a constant finding, and often anuria occurs.

Uremia, coma and death follow from four to fifteen days after the transfusion.

2. In contradistinction, according to the same authors, "Hemolysis, hemoglobinuria and anuria are absent" in anaphylactic or proteolytic reaction due to the presence in the donor's blood serum of nonspecific proteins and other substances which when transfused may "elicit . . .

51. DeGowin, E. L.: Grave Sequelae of Blood Transfusions: Survey of Three Thousand and Five Hundred Cases, Ann. Int. Med. **11**:1777, 1938.

an allergic repsonse from the recipient." Results of adverse reactions which develop during transfusion or shortly afterward are "high fever, asthma from intense bronchiolar spasm, involuntary evacuation . . . and death in a few hours from asphyxia."

3. A survey of hospitals using the citrate method exclusively indicates that these institutions reported either few or no reactions or else

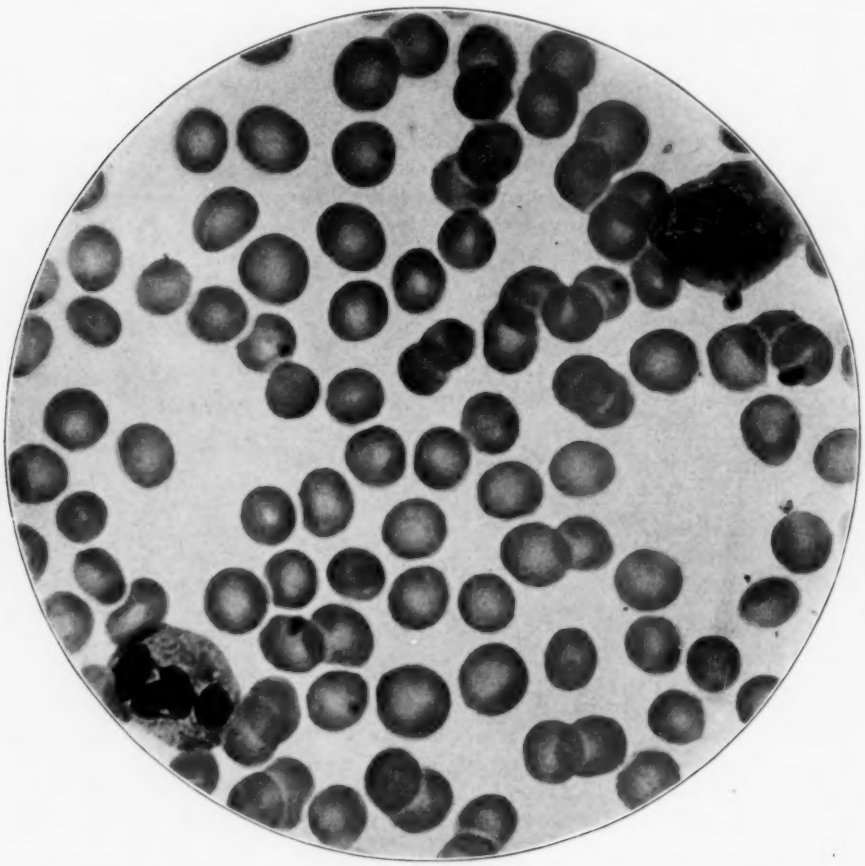

Fig. 1.—Normal distribution of red cells and mature neutrophilic granulocytes, the latter showing a uniform pattern of the chromatin of the nuclei and cytoplasm packed with small granules of uniform size. The normal stimulus for the formation of new granulocytes to replace those lost by cell death in the circulation or in the tissues seems to be due to substances released when the nuclei disintegrate. (Department of hematology, Harper Hospital.)

a relatively high incidence. This difference may perhaps be explained by the varying care taken in preparing the transfusion apparatus and the quality of the citrate solution employed. The meticulous care

required for the successful use of the citrate method was described in detail in the paper of Lewisohn and Rosenthal.[52] Some authors found it best not to use this method in cases of extreme shock or profound states of anemia, in which conditions the extra hazard should be avoided by the use of whole unmodified blood. Others have charged

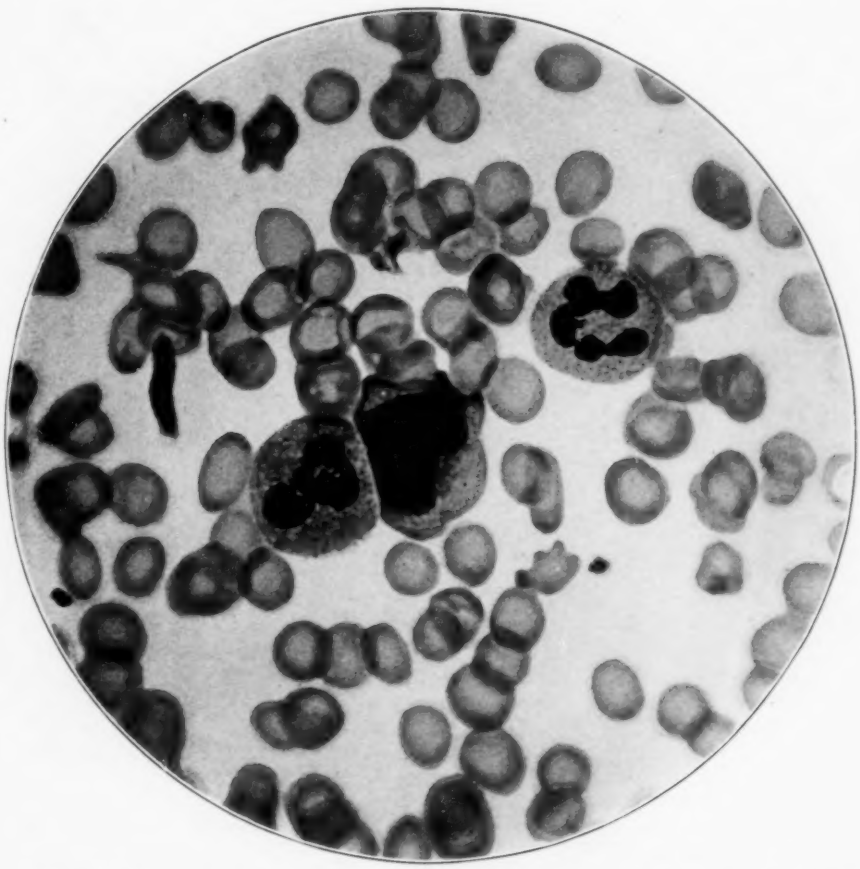

Fig. 2.—Hypochromic anemia in severe sepsis; hemoglobin content reduced to 36 per cent; red blood cell count, 2,800,000. One stab cell shows nuclear pyknosis. (Department of hematology, Harper Hospital.)

also that citrate destroys platelets, that it develops anticomplementary properties in plasma, that it reduces the phagocytic and opsonic index of the blood, that the red blood corpuscles are made more friable and

52. Lewisohn, R., and Rosenthal, N.: Prevention of Chills Following Transfusion of Citrated Blood, J. A. M. A. **100**:466 (Feb. 18) 1933.

that it produces a general systemic reaction with malaise and chills. Polayes and Lederer [48] reported that

> Lederer, in a comparative study of a consecutive series of 100 transfusions of citrated and whole blood transfusions, encountered about 50 per cent reactions with citrated and none with whole blood transfusions. . . .

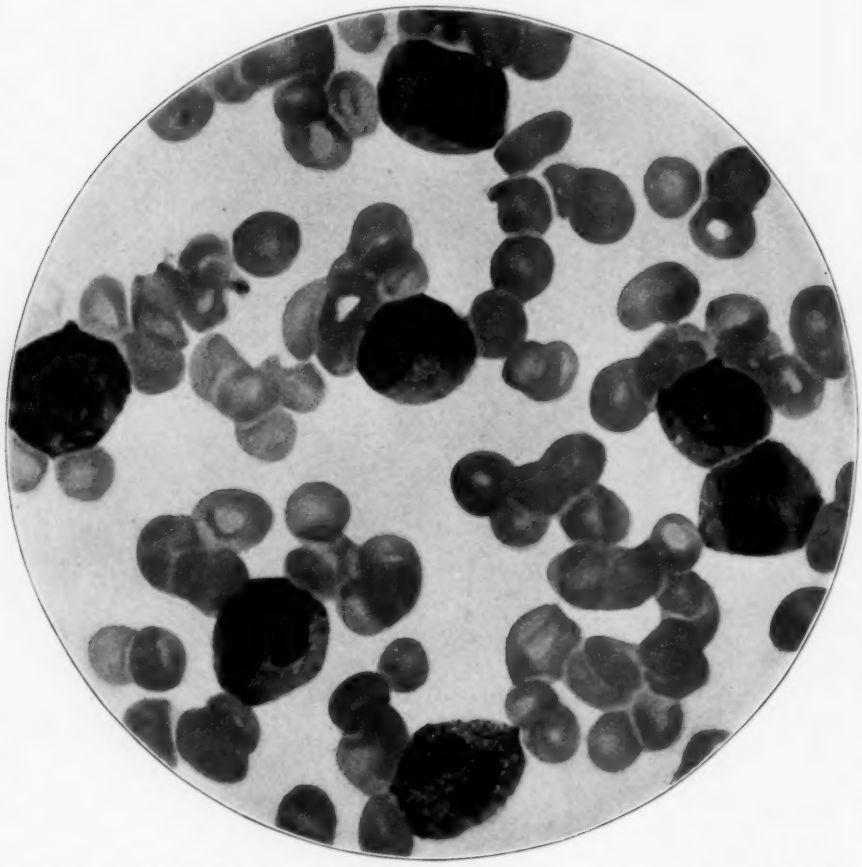

Fig. 3.—Peripheral blood in sepsis and toxemia; marked shift to the left; characteristic degenerative and regenerative changes; cytoplasm decreased and containing coarse "toxic" granulations; marked leukocytosis. (Department of hematology, Harper Hospital.)

> The exact causes for these reactions are still unknown . . . [but are believed by some to be] due to the prolonged exposure of the blood to foreign substances that the citrate method necessitates. . . . [others have concluded] that citrate produces certain chemical and biologic changes in the blood, which changes may account for the reactions. Whichever is the correct explanation,

it is quite obvious from the review of the literature, that reactions are much more common with citrated than with unmodified blood transfusion.

COMMENT AND SUMMARY

Irrelevant to controversial political trends in medical practice, there are beneficial trends in medical investigation which none can deny.

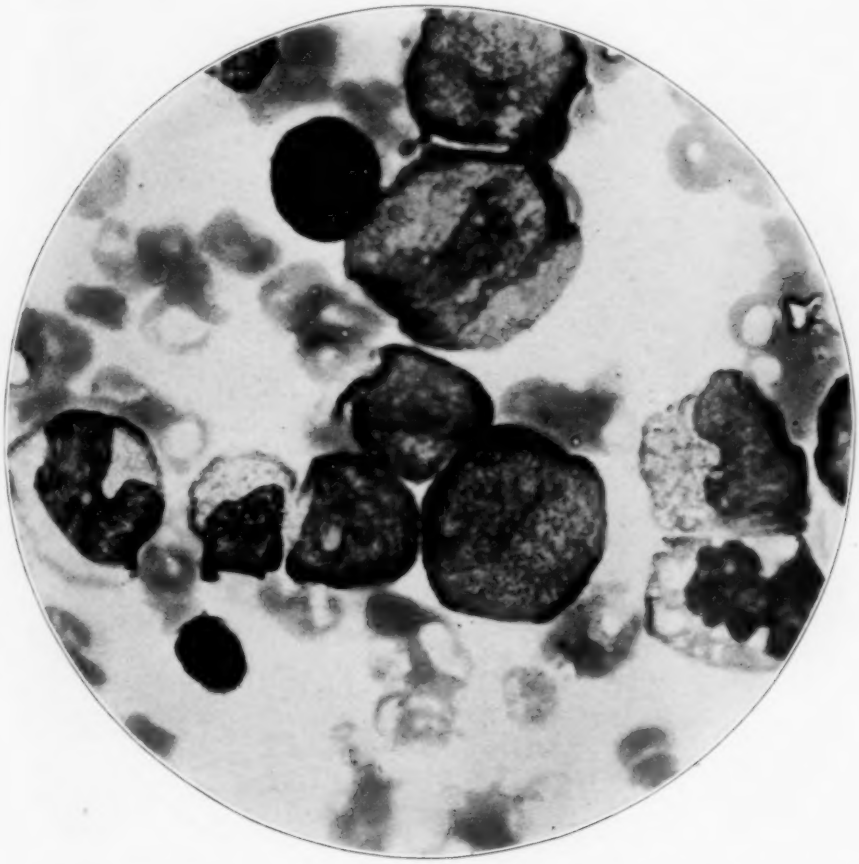

Fig. 4.—Effects of toxemia on bone marrow. Coarse basophilic granulation of neutrophils; vacuolation of the cytoplasm; pyknotic nuclei; high concentration of "stab" forms, myelocytes and premyelocytes. All the cells show characteristic degenerative changes. (Department of hematology, Harper Hospital.)

The change in the attitudes and standards of medical therapeusis, particularly noted within the last two decades, is evidenced in the field of otolaryngology as follows:

1. By an increasing cognizance on the part of the otolaryngologist, the bacteriologist, the hematologist, the internist, the pathologist and the

pediatrician that their ultimate objective, the diagnosis and treatment of disease, can be attained only by their collaboration.

2. By a new respect on the part of the surgeon for the benefits to be derived from efficient application of biologic and chemical therapy as supplements to the surgical drainage of infected foci.

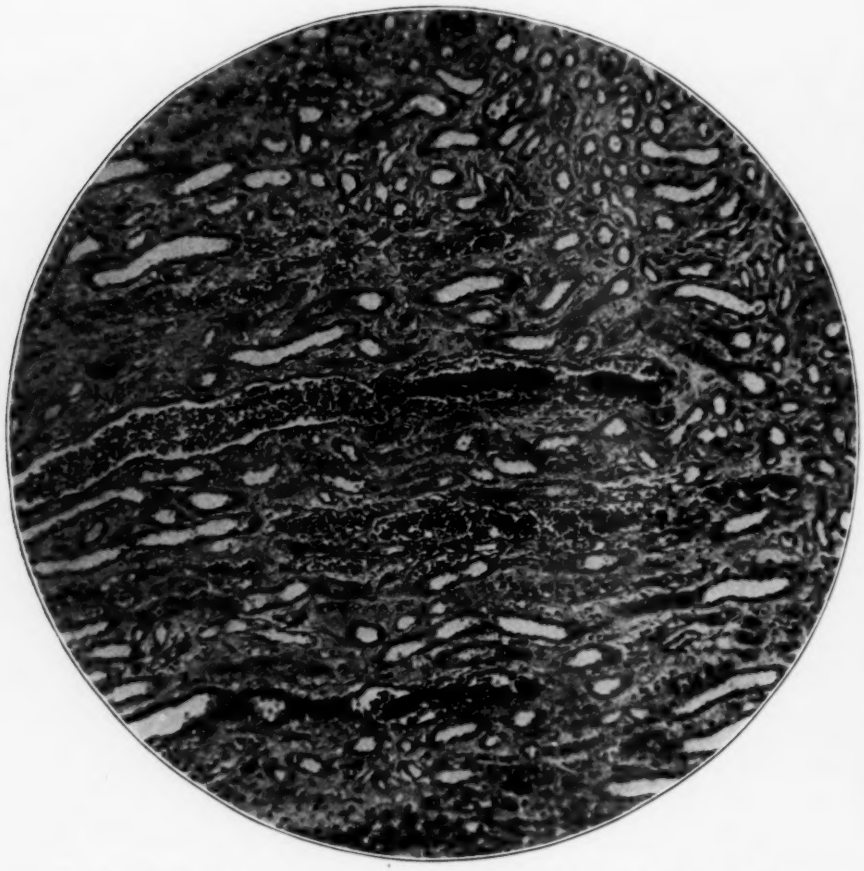

Fig. 5.—Transfusion death. Photomicrograph of the kidney showing acute tubular hemorrhages. Some of the lesions are due to the hemolysis and some to the agglutination of red corpuscles; blood pigment and disintegrated leukocytes are present. (Courtesy of Dr. Plinn W. Morse, pathologist, Harper Hospital.)

3. By recognition of the fact that statistical figures on infectious conditions must be broken up according to age groups and types and strains of the infecting organism and allowances must be made for

yearly or seasonal variation and for the "unrecognized variables" which complicate all clinical investigations.

4. By awareness of the need for a compilation from clinical records of careful objective observations, correlated with the correct interpretation of quantitative and qualitative changes in the blood and other laboratory findings, the analyses of which depend on a thorough understanding of the physiologic principles involved.

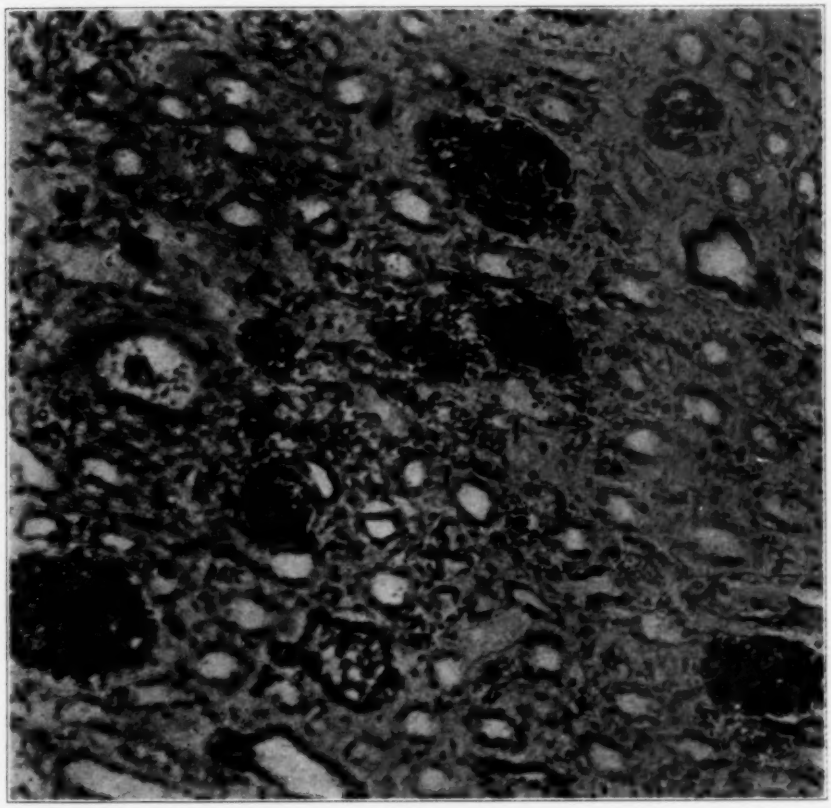

Fig. 6.—Transfusion death. Photomicrograph of the kidney showing more severe tubular involvement than in figure 5. Anuria may be due to blocking of the tubules by blood pigment. The kidneys in these cases are swollen and reddish brown. (Courtesy of Dr. Plinn W. Morse, pathologist, Harper Hospital.)

CONCLUSIONS

From a comparative study of the data presented and an examination of the photomicrographs showing the characteristic changes in blood and bone marrow which take place during the course of otitic

sepsis and the fatal renal hemorrhages resulting from the transfusion of incompatible blood, one may formulate the following conclusions:

To influence or control effectively the activities of the individual types of cells in blood and tissues, one must acquire specific information concerning their fundamental physiologic relations, not only to each other but to the other cells and functions of the body.

The effectiveness of preoperative or postoperative transfusions of blood in ameliorating or controlling the adverse activities of blood, bone marrow and parenchymal organs in otitic sepsis, is not dependent on the choice of the direct or the indirect method of intravenous injection. Their efficiency depends on their fulfilling the purpose for which they may be given: to replace erythrocytes lost in anemia, to restore the level of plasma protein, hemoglobin and oxygen carriers, to counteract leukopenia (in rare cases) and/or to increase the blood volume and water balance, thereby diluting toxic blood.

The use of immunotransfusion is different in purpose, in that its therapeutic value depends on the antibodies and other immune substances contained in the donor's blood, rather than on the blood elements themselves. Type specificity of the various strains of organisms encountered in otitic sepsis complicates the question of immunity. This is particularly evidenced in efforts to compile clinical data correlated with bacteriologic investigations of the biologic behavior and antigenic structure of the troublesome triumvirate, Streptococcus group A, Pneumococcus type III and Staph. aureus. These efforts may culminate, in the near future, in the establishment at convenient centers of a cooperative service of direct specific biologic therapy.

Irrespective of the type of blood to be transfused or the method of its administration, the transfusion of blood should not be considered a casual routine procedure. With definite indications for its use, the value of blood therapy for otitic sepsis may be greatly enhanced by administering it at the earliest indication that it is needed and by repeating it as often as it is required while the recipient's defense forces are capable of responding and before sepsis or toxemia or both are overwhelming.

CHEMOTHERAPY

THOMAS C. GALLOWAY, M.D.
EVANSTON, ILL.

The emphasis of this symposium seems wisely placed. What happens after the primary operation usually determines whether the patient lives or dies and whether he regains a well healed ear and mastoid with normal function or has a persistent pathologic condition. It is the complications of otitis and mastoiditis that after all determine the mortality. The greatest factor in controlling these complications seems to me to be chemotherapy.

A brief general review of the use of sulfanilamide and allied compounds seems to be necessary to an understanding of the full possibilities of postoperative care. Many papers have discussed these agents, and Fenton [1] and McLaurin [2] covered the fundamentals before sections of the American Laryngological, Rhinological and Otological Society. Schenck [3] gave an excellent summary of over two hundred and fifty titles which had appeared up to November 1938, but so much new knowledge is constantly being reported that it is difficult to keep up with the subject.

For many years efforts had been made for the chemical control of otogenic sepsis but almost without avail. Some promise had been given by substances like Pregl's iodine solution and ethylhydrocupreine given after the technic of Kolmer and Madden [4] by intracarotid injection, but few real results were obtained. A few other chemicals, like mercurochrome given intravenously, seemed to give some help, but Mellon stated the belief that this was only protein shock therapy.

Then came the group of compounds that have brought an entirely new outlook in the fight against grave otogenic infections. They have

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Read as part of the Symposium on the Care of the Patient after Operations for Sepsis of Otitic Origin at the Forty-Fifth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., Chicago, May 9, 1939.

1. Fenton, R. A.: Sulfanilamide in Otolaryngology, Ann. Otol., Rhin. & Laryng. **48**:17 (March) 1939.

2. McLaurin, J. G.: Sulfanilamide in Otolaryngology, Ann. Otol., Rhin. & Laryng. **48**:23 (March) 1939.

3. Schenck, H. P.: Use of Sulfanilamide in Otolaryngology, Arch. Otolaryng. **28**:698 (Nov.) 1938. (This is an excellent summary of the literature.)

4. Kolmer, J. A., and Madden, B.: Chemotherapy and Serum Therapy of Pneumococcus and Streptococcus Meningitis: Intracarotid Treatment for Experimental Pneumococcus Meningitis, Arch. Otolaryng. **9**:509 (May) 1929.

happily modified the whole prognosis for seriously ill patients and at the same time have markedly changed the practice of surgical otology. Probably this will lead, as progress usually has, to a considerable degree of technological unemployment in hard acquired skills. Compensating for this must be the great satisfaction of being able to cure disease before which one previously stood helpless. Also, new problems have been raised that will require the utmost experience and training, and, while it may not so often be employed, the same surgical judgment and facility in operative technic must still be available.

To visualize what has happened a few statistics will be cited. In 1935 at the Evanston Hospital there were 37 cases of acute mastoiditis requiring surgical intervention, with 5 deaths. In 1938 there were 5 such cases, with no deaths. In the community served by this hospital in the last two years sulfanilamide has been given increasingly at or even before the onset of otitis. In the same metropolitan area, at Cook County Hospital there was no marked decrease in the incidence of mastoiditis requiring surgical intervention, though the number of deaths was substantially reduced and 9 patients with streptococcic otitic meningitis recovered, whereas none, so far as I know, had previously recovered. The patients at this institution came from a group with little medical supervision previous to coming to the hospital, with the surgical condition in most cases well advanced. In other words, as virulent infection as usual was present in the community, but when sulfanilamide was used early and fully the control of the infection was striking.

Horan and French [5] found the percentage of mastoiditis complicating otitis reduced from 23 to 4.5 in an English naval hospital. Though more conservatism may have been practiced because of confidence in the drugs, this controlled series seems convincing. Campbell [6] at Ottawa General Hospital found the days in the hospital for patients with acute otitis reduced from an average of twenty-four to eight under conditions similar except for the use of sulfanilamide. In addition many more recoveries from otitic meningitis have been reported in the few years since these drugs have been used than altogether previously. At Cook County Hospital the last 5 consecutive patients with proved bacterial otogenic meningitis have recovered with chemotherapy.

Only the more definitely established facts concerning the most important chemical agents will be given here. Sulfanilamide seems to be the outstanding preparation. Much work has been done to determine its mode of action, yet there is complete agreement only on the fact that it is

5. Horan, V. G., and French, S. G.: Sulfonamide in the Treatment of Acute Mastoiditis, Brit. M. J. **2**:2519 (Dec. 31) 1939.

6. Campbell, G. A.: Otitis Media from the Pediatrician's Viewpoint, Canad. M. A. J. **40**:146 (Feb.) 1939.

bacteriostatic. When it is used in vitro or in vivo, susceptible bacteria cease to multiply—in the latter case, after a lag of several hours to allow for absorption.

This may be due to a direct toxic effect on the organism, but Whitby [7] suggested that the effect is an alteration of the ability of the bacteria to assimilate nourishment, possibly by blocking of a receptor mechanism. Levaditi [8] suggested that the action is due to a combination with protein of the host, especially with capsulogenous material. Locke, Main and Mellon [9] suggested that the accumulation of peroxide, to which pneumococci and streptococci are sensitive, may explain the action of sulfanilamide. Normally this peroxide is produced by them but is decomposed by a catalase. This catalase can be inactivated by substances arising from sulfanilamide under certain conditions. Mellon stated also that organisms may be changed by these agents from a virulent mucous phase to an avirulent rough one, such change persisting for some generations, though the process may be reversible. From his experimental work, Mellon [10] stated the belief that there is a marked potentiation by sulfanilamide of the immunologic effect, that is, an enhancement of the effect somewhat like synergism, by which the result is several fold the expected additive effect.

With cessation of the organisms' growth, it still remains for them to be destroyed, and the defense reactions of the body then operate normally but without the deterrent effect of bacterial toxins.

Osgood [11] stated the belief, based on his experiments with marrow culture, that the toxins, the aggressins and the hemolysins are neutralized or diverted, but Long, Bliss and Feinstone [12] offered the simple explanation that in the absence of multiplying organisms the formation of toxin is negligible. Phagocytosis does not appear to be specifically stimulated, but in the absence of negative chemotaxis and toxins the leukocytes and macrophages engulf and destroy the organisms

7. Whitby, L.: Chemo-Therapy of Bacterial Infections, Lancet **2**:1095 (Nov. 12) 1938.

8. Levaditi, C., cited by Mellon, Gross and Cooper,[10] p. 227.

9. Locke, A.; Main, E. R., and Mellon, R. R.: Anti-Catalase and the Mechanism of Sulfanilamide Action, Science **88**:620 (Dec. 30) 1938.

10. Mellon, R. R., in Mellon, R. R.; Gross, P., and Cooper, F. B.: Sulfanilamide Therapy of Bacterial Infections, Springfield, Ill., Charles C. Thomas, Publisher, 1938.

11. Osgood, E. E.: Culture of Human Marrow: Studies on the Mode of Action of Sulfanilamide, J. A. M. A. **110**:349 (Jan. 29) 1938. Osgood, E. E., and Brownlee, I. E.: Mode of Action of Sulfanilamide, ibid. **110**:1770 (May 21) 1938.

12. Long, P. H.; Bliss, E. A., and Feinstone, W. H.: Mode of Action, Clinical Use and Toxic Manifestations of Sulfanilamide, J. A. M. A. **112**:115 (Jan. 14) 1939.

in the blood, fluids or tissues where phagocytes can penetrate. The spread of the disease locally may then be sharply halted, as shown histologically by Mellon.[10]

Locke [12a] has emphasized that the condition of the host is an important factor and has shown in experimental animals that the presence of antibodies is necessary and that other accessories, as vitamins B_1 and C and the hormone of the adrenal cortex, are of much value. Especially must opsonins and other immune bodies be present, and if there is any doubt they must be supplied by blood transfusions or convalescent serum and in the case of the pneumococcus by type specific serums of the horse or rabbit.

Sulfanilamide is absorbed rapidly and within four hours may reach a therapeutic concentration in the blood. It also quickly reaches from 50 to 75 per cent of the same concentration in spinal fluid, exudates, transudates, empyemic fluid, pleural fluid, urine and even fetal blood.[13] The ability to absorb and hold effective concentrations in contact with organisms in pathologic processes seems to be the factor which determines its effectiveness. Kopetzky [14] expressed the opinion that bacteria invading the temporal bone may be protected from the drug. I have found persistently positive cultures provided from a walled-off area of coalescence in the tip of a mastoid otherwise healed with sulfanilamide. Lockwood [15] stated the belief that peptone-like products in pus and in tissue undergoing necrosis prevent the proper effect of sulfanilamide on infections.

Sulfanilamide is most effective against the beta hemolytic streptococcus, but Hoare [16] showed by experiments in vitro, along with clinical studies, that some strains of this group may be resistant, and it is a matter of common observation that not all strains respond equally well. Pneumococci are affected less by this drug and green-forming streptococci much less. Bacillus influenzae is moderately susceptible.

The development of strains resistant to the drug may be of some importance, as suggested by Whitby.[7] Mellon [10] expressed the opinion that this may occur and that strains which are incompletely destroyed may be changed to another phase, remain quiescent for a time and then be insusceptible to sulfanilamide. Long expressed doubt of this.

12a. Locke, A., cited by Mellon,[10] p. 281.

13. Marshall, E. K., Jr.; Emerson, K., Jr., and Cutting, W. C.: Para-Aminobenzenesulfonamide: Absorption and Excretion: Method of Determination in Urine and Blood, J. A. M. A. **108**:953 (March 20) 1937.

14. Kopetzky, S. J.: The Management and Treatment of Otogenic Meningitis, Ann. Otol., Rhin. & Laryng. **47**:117 (March) 1938.

15. Lockwood, J. S.: Observations on the Mode of Action of Sulfanilamide and Its Application to Surgical Infections, Ann. Surg. **108**:801 (Nov.) 1938.

16. Hoare, E. D.: Bactericidal Changes Induced in Human Blood and Serum by Sulphamido-Chrysoidine and Sulphanilamide, Lancet **1**:655 (March 19) 1938.

Repeatedly I have observed cases in which administration of the drug was discontinued too soon or a primary focus was not completely removed and in which the patient responded as well to a second course as to the first.

The dosage of the drug seems to have been rather well standardized. Long, Bliss and Feinstone [12] presented a table which gives the usual practice. My associates and I have usually given about ¾ of a grain (0.05 Gm.) per pound (0.5 Kg.) of body weight per day for three days and ½ grain (0.03 Gm.) subsequently. Slightly larger doses may be given for more virulent infection and to children, who seem to tolerate the drug better. Smaller doses suffice for less severe infections, although some observers believe the forms most virulent clinically are most susceptible to the drug. The initial doses should be large in order rapidly to reach an effective concentration in the blood, and the later doses should be distributed at four hour intervals to maintain that concentration. Covering doses of sodium bicarbonate have been advised to help to prevent acidosis.

The drug is rather rapidly excreted by the urine, and poor renal function may lead to an excessive concentration in the blood. Conversely, an excessive intake of fluids may cause too rapid elimination of the drug, though that may be desirable if toxic effects occur. Long, Bliss and Feinstone [12] advised limiting fluids to 2,500 cc. per day in the average case. The dosage should be gaged by determinations of the level of sulfanilamide in the blood, and it is advised to maintain a level of between 5 and 10 mg. per hundred cubic centimeters. The therapeutic effect does not always parallel the concentration, and I have seen excellent results in cases with a level of 2 mg. per hundred cubic centimeters. Mellon cured one of his early patients with streptococcic meningitis with 16 grains (1.04 Gm.) of the drug given over three days, and Osgood [11] on the basis of his experiments with marrow stated the belief that concentrations of 1 : 100,000 to 1 : 10,000 of sulfanilamide are effective. Further experience may revise ideas of dosage.

Ordinarily the drug is to be given by mouth, and nothing seems to be gained in rapidity of effect by parenteral administration. Intrathecal administration has not been shown to give added benefit. If nausea or gastric distress make oral administration impossible, sulfanilamide may be given in 0.8 per cent solution in lactate-Ringer (Hartman's) or saline solution by hypodermoclysis. In a small series my associates and I felt that we got more toxic effects this way.

Toxic effects do occur, but in most cases are not very important. Cyanosis is common and apparently without much significance, as are nausea, vomiting, dizziness, headache and confusion, and these symptoms ordinarily do not require that administration of the drug should be discontinued. Fever occurs in about 5 per cent of cases, and Long,

regarding it as a warning against graver toxic effects, advised discontinuing administration of the drug when it occurs. If the rise in temperature is of 1 or 2 degrees (F.) only, that has hardly seemed necessary when the drug was badly needed. Rash is among the more serious signs. The hemoglobin content is reduced as a rule from 10 to 20 per cent as is the proportion of white cells. Severe granulocytopenia has occurred, usually without much warning, but has occurred rarely except when the drug has been given for more than sixteen days to a total amount of 30 to 60 Gm. Ten deaths have been reported from this complication, which is a small price to pay for the protection that has been given.[17] Acute hemolytic anemia and jaundice are of grave importance, and usually indicate that administration of the drug should be discontinued. In acute hemolytic anemia there may be preliminary leukocytosis with a rapid fall in the hemoglobin content. Transfusions may be required if it occurs, but for serious conditions, as, for instance, meningitis, administration of the drug may even be continued, if the blood is kept toward normal by repeated transfusions. Erythrocyte and leukocyte counts and determinations of the hemoglobin content should be done every two days.

Whether one discontinues administration of sulfanilamide in the presence of toxic effects depends on their nature and their severity and on the virulence of the infection. If there is apparently the question of avoiding simple mastoidectomy or not in the presence of acute hemolytic anemia or hyperpyrexia, it seems wise to discontinue administration of the drug. If unconquered meningitis is present, administration of the drug should be kept up, complications being met as they arise.

Sulfanilamide has been discussed as the typical member of this series. Hundreds of related compounds have been investigated, and it is likely that some better drug will be evolved. At present sulfanilamide appears to be the leader by virtue of its relatively simple constitution, stability, fairly uniform action, high solubility, good absorption and rather low toxicity.

Neoprontosil, or prontosil soluble, was widely used before its chief activity was found to be probably due to freeing the sulfonamide radical in the body. This drug is inactive against streptococci in the test tube, and most investigators believe that its effect in the blood is due only to the simpler radical split from it. Domagk, who did the original work with prontosil, and Bannick, Brown and Foster,[18] of the Mayo clinic, stated the belief, however, that it has some additional action and that

17. Carr, P., and Root, R. N.: Granulocytopenia, J. A. M. A. **112**:1939 (May 13) 1939.

18. Bannick, E. G.; Brown, A. E., and Foster, F. P.: Therapeutic Effectiveness and Toxicity of Sulfanilamide and Several Related Compounds: Further Clinical Observations, J. A. M. A. **11**:770 (Aug. 27) 1938.

it is more effective than sulfanilamide and less toxic. Osgood suggested that it may be more effective with a lower concentration in the blood because of its slower breakdown with a constant, though minimal, effective dose in the blood. It is of value given intramuscularly when sulfanilamide cannot be taken by mouth and seems definitely to be tolerated in some cases in which sulfanilamide can no longer be taken.

An increasing number of reports testify to the value of sulfapyridine. It is claimed to be as efficacious against the beta hemolytic streptococcus as is sulfanilamide, though Long denied this, and has much greater effect against pneumococci, with some protection against Streptococcus viridans, Friedlander's bacillus, B. influenzae, and staphylococci. Some work indicates that it injures the protective capsule of the pneumococcus.[7] This drug is less soluble and has a varying rate of absorption, with some variation in the clinical effects. Whitby [7] stated the belief that it is less toxic than sulfanilamide, but Long [19] explained the diminished toxicity on the basis of lower solubility and poorer absorption and maintained that it has no advantage when the amount actually absorbed is taken into consideration. Marshall, Bratton and Litchfield [20] found the soluble sodium salt quite as toxic as sulfanilamide. My patients have seemed to find sulfapyridine nauseating, and gastric distress and mental depression have seriously lowered morale in a number of cases.

Some special questions require discussion. Among these are the following: How do these drugs modify pathologic conditions? How do they modify symptoms? When is surgical intervention or additional surgical intervention needed with them? Do all foci need drainage or removal?

The modification of pathologic conditions depends on the vulnerability and the accessibility to the drug of the infecting organism and the time of administration. Mellon showed that in inoculated foci in experimental animals the spread of disease may quickly be arrested by administration of sulfanilamide, with rapid healing. From my experience with operations on the mastoid when sulfanilamide had been thought to have failed, I think that complete recovery can take place in spite of extensive involvement. For obvious reasons this statement must not be interpreted as advising expectant treatment in such cases. In cases of mastoiditis in which the drug has been given fairly early, at operation one is likely to find a spotty pathologic condition. Areas of nearly normal hard bone may alternate with zones where the bone is soft and areas of

19. Long, P. H.: Sulfapyridine, J. A. M. A. **112**:538 (Feb. 11) 1939.

20. Marshall, E. K., Jr.; Bratton, A. C., and Litchfield, J. T., Jr.: The Toxicity and Absorption of 2-Sulfanilamidopyridine and Its Soluble Sodium Salt, Science **88**:597 (Dec. 23) 1938.

previous breaking down may be filled with granulations varying from soft to firm, organizing ones, with some cells even containing pus, with or without culturable organisms. In a mastoid operated on in the fifth week, chiefly on account of pain, there had been extensive destruction, yet no pus was found, culture was negative and granulations were everywhere firm and fibrous. This, I think, would have completely healed without intervention. In another case the patient had had stormy serous labyrinthitis and elevation of temperature to 99.5 F. persisting in the fifth week, with a moderate discharge showing numerous colonies of hemolytic streptococcus even after full doses of sulfanilamide. At operation a cavity 1.5 cm. wide in the tip was found filled with pus. This might, I think, have continued almost indefinitely. A section from a mastoid of a patient for whom administration of sulfanilamide had to be stopped on account of acute hemolytic anemia showed considerable definite formation of new bone with a matrix, osteoblasts and recent calcification.

Mellon and Whitby [7] expressed the opinion that these agents do not modify the immune reactions; Long [21] stated the belief that they may. It seems that if the compounds are given early there may be not enough general and local damage to provoke the reactions of immunity. Some observers [22] have noted that when convalescent serum and sulfanilamide were used early for scarlet fever, the Dick reaction remained positive after recovery. It has seemed to me that in a few cases in which the drug was given early and administration then had to be stopped on account of toxic effects, the patient progressed as one who only then began to develop local and general immunity—that is, that the age of the disease was dated approximately from the time when administration of the drug was discontinued.

It is important to know how symptoms can be modified by these drugs, because modified criteria of diagnosis must be established if administration of the drug is to be continued for its best effect when complications are imminent and not discontinued, as seems too commonly done, for fear of masking symptoms. Symptoms of sepsis may be either simulated or suppressed.

Simulated symptoms due to the drugs may be fever, with which the temperature may vary from ½ to 4 degrees (F.) above normal and which may require that administration of the drug should be stopped at least temporarily for clarification, although lack of leukocytosis is an important differentiation; nausea and vomiting, which according to the belief stated by Long are due to central irritation; cyanosis and depression, which might be taken for the appearance of sepsis but usually

21. Long, P. H.: Sulfapyridine, J. A. M. A. **112**:1850 (May 6) 1939.
22. Sauer, L. W.: Personal communication to the author.

can be recognized as quite different; headache and neuritic pains, which in several cases were quite like those of meningeal irritation, and depression and confusion, which are not always easy to distinguish from toxic cerebral depression or encephalitis (chart 2).

Suppression of symptoms probably occurs only because the activity of the organisms is inhibited. Long, for instance, showed that sulfanilamide had no antipyretic effect in typhoid fever. As an example of

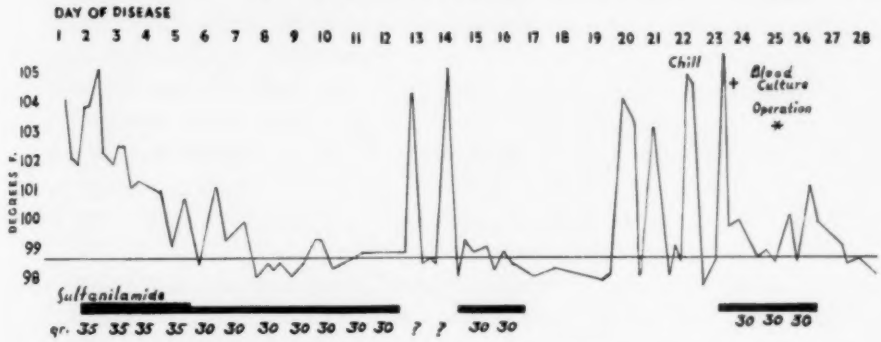

Chart 1.—Masked sinus thrombosis. At the point designated ? ? the patient vomited and little or none of the drug was absorbed. At operation the sigmoid sinus was found filled with a septic clot.

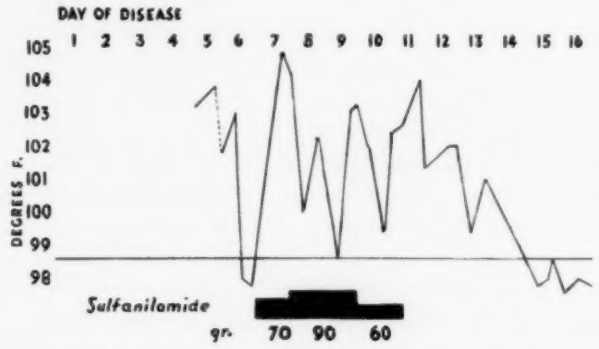

Chart 2.—Simulated encephalitis. The patient had hyperpyrexia, severe headache, nystagmus and asynergy and was disoriented. The picture suggested encephalitis but cleared within forty-eight hours after the administration of sulfanilamide was stopped.

suppressing grave sepsis, the steeple temperature curve in a case of sinus thrombosis twice flattened to normal when sulfanilamide was given, although at operation the sinus was found filled with a septic clot (chart 1).

23. Footnote deleted.

The diagnosis of complications in the presence of chemotherapy may be difficult, but with experience with these agents it has seemed that seldom in the individual case is one unable to ascribe to the drug and to the disease their proper places. If in spite of adequate dosage of the drug important symptoms persist, the need of intervention is still greater than when symptoms are present without the drug, and operative indications must be modified accordingly.

The question of the masking effect seems to be handled quite differently by various authors by whom it has been discussed. Statistical evidence already quoted seems to me to support the opinion that for all threatening otitis the drugs should be given, and, whether otologists agree or not, the pediatricians seem to have decided the question this way. To withhold it at the threatening stage for fear of masking the disease appears no more logical than for a fireman to turn off his hose to see what a fire is going to do. On the other hand, many authors believe that if sulfanilamide is to be effective, results will be definite in from three to five days or perhaps seven. If no serious trouble develops and if it appears to have some favorable effect, administration of sulfanilamide should probably be continued still longer. If then the clinical picture is not clear and the condition still not serious, administration of the drug should be discontinued in order to show the true manifestations of the disease. Fever is most likely to complicate the picture, and for patients otherwise doing well it is usually safe to stop administration of the drug and postpone operation for two days to be sure the chemical is not responsible. Likewise if active disease seems to be cured, especially a deep process, it is generally advised to continue administration of the drug for four to six days more and to observe the patient closely for some time thereafter. Lockwood [15] and others have shown that pocketed processes may be apparently inactive but recrudesce after the drug is eliminated.

It is obvious that the classic indications for operation should hold if the condition is seen fully developed or if it is necessary to discontinue administration of the drug on account of its toxic effects. When the drug has not been given, it is usually safe in the absence of the more serious complications to administer it and wait, as spread of the process is then less likely. I have seen surprising recoveries occur in cases of marked tenderness, swelling, roentgen evidence of softening of the bone and even subperiosteal abscess.

There will also be less necessity for early intervention even in the virulent infections with hemolytic streptococci, as these seem to be the organisms most vulnerable to sulfanilamide. Even the threat of complications probably should not hasten intervention within the first week of onset, as such infections have repeatedly been controlled by the

drug. In a case previously reported from Cook County Hospital, hemolytic streptococcic meningitis developed five days after the onset of acute otitis. Discharge from the ear was hemorrhagic and profuse. At the suggestion of Dr. S. J. Pearlman, operation was not done, since presumably the path of infection was through vascular channels. The meningitis speedily cleared under sulfanilamide therapy. The ear stopped discharging, and operation was not necessary. In a second case of meningitis on the sixth day of otitis similar treatment brought about recovery. Such examples, however, should not lead to failure to remove all active foci.

Also the nature of the operative intervention may be changed when it is apparent that sulfanilamide is controlling infection. One may be more conservative, as suggested by Kopetzky, in discussing a case of acute labyrinthitis in which he found it unnecessary to open the labyrinth. Some surgeons may have less respect for the dural barrier, though that may prove deplorable, but a certain protection may be afforded when it appears necessary to open the dura.

That all diagnosed foci of active disease should be removed or drained, even when the infection is apparently controlled by the drug, seems generally agreed. It seems much wiser, at least with the more serious complication of meningitis, sinus thrombosis or abscess of the brain to leave no possible active focus which is amenable to reasonable surgical intervention.

Meningitis if due to the hemolytic streptococcus seems to be well treated by the usual administration of the drug, with spinal drainage as indicated by the symptoms. In reporting 6 recoveries from pneumococcic meningitis, Finland, Brown and Raub [24] seemed to have an improved technic. Sulfanilamide was given in full doses by mouth and then type specific serum by vein. After two hours, blood was withdrawn, the serum separated and 5 to 10 cc. of this serum given intraspinally in order to supply the complement, which is lacking in the spinal fluid. Rhoads, Horswell and Fox [25] were unable to duplicate this success with the same method, but had 5 recoveries in 17 cases with the combined use of serum and sulfanilamide.

Overenthusiasm for the drugs has already led to some disasters. They cannot replace training, knowledge and care in medical practice, and their limitations must constantly be borne in mind. There are numerous reasons why they may fail.

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24. Finland, M.; Brown, J. W., and Raub, A. E.: Treatment of Pneumococcic Meningitis, New England J. Med. **218**:1033 (June 23) 1928.

25. Rhoads, P. S.; Horswell, R., and Fox, W.: Treatment of Pneumococcic Meningitis with Specific Antipneumococcic Serum Plus Sulfanilamide or Sulfapyridine, to be published.

Certain organisms, as the staphylococcus and Str. viridans, seem hardly at all susceptible to them. The presence of these organisms in mixed culture apparently prevents the full effect of the drug on susceptible organisms. Also resistant strains of ordinarily vulnerable bacteria may occur. Hoare, for instance, found little clinical response to sulfanilamide in some cases of puerperal sepsis due to hemolytic streptococci and demonstrated in the laboratory that these strains were refractory.

Protected foci, or dead spaces to which drugs do not penetrate, were postulated by Ballenger, Elder, McDonald and Coleman,[26] Kopetzky [14] and others as a cause of failure. One sees such pockets not infrequently in an otherwise healed mastoid. Lockwood [15] found that peptone and peptone-like bodies rendered sulfanilamide relatively ineffective and stated the belief that their presence in pus or tissue undergoing necrosis explains some failures.

Other causes of failure reside, as Mellon [10] and his co-workers showed, in general factors pertaining to the host. Lack of opsonins, of the complement and of other immune bodies, anemia, lack of phagocytes, chilling, general debility, deficiency of vitamins and other factors can prevent cure in spite of the bacteriostatic effects of the drug.

Toxic effects also may prevent realizing the beneficial effects of the drugs. Improper administration is an important reason for failure. Infrequent spacing may allow the concentration, especially of the more soluble drug, sulfanilamide, to fall below the effective level. Osgood stated the belief that the maintenance of this minimal level is most important. Failure to retain or absorb the drugs is frequently overlooked, as it would not be if frequent determinations of the level in the blood were made, and the vomiting of the drugs before absorption also is not always taken into account. If cultures are not taken, a nonsusceptible organism may be futilely treated. Alertness for possible serious complications can easily fail and proper consultation not be sought until too late, especially if there is overconfidence in the drugs. Herein lies grave danger.

On the basis of the foregoing observations, certain principles may be formulated for postoperative chemotherapy as a ground for further discussion. Treatment in each case must, of course, be individualized.

If chemotherapy has not been administered before operation and the patient presents fully developed indications for intervention, with a presumably susceptible organism, it seems that the drug should be given

26. Ballenger, E. G.; Elder, O. F.; McDonald, H. P., and Coleman, R. C., Jr.: Failures in theTreatment of Urinary Tract Infections with Sulfanilamide, J. A. M. A. **112**:1569 (April 22) 1939.

to shorten convalescence, safeguard against complications and diminish mortality. Masking of more serious trouble should be borne in mind.

If in spite of presumably adequate chemotherapy operation becomes necessary, further administration of a drug is not needed if the disease is removed or the area satisfactorily drained. Usually, however, the drugs will have had some effect, and failure may have been due to too late or improper administration, so that, if further trouble threatens, the drugs still seem desirable if they are well borne.

If toxic symptoms occur, especially the more severe ones, and surgical intervention seems sufficient, administration of the chemicals is stopped. If meningitis develops, probably administration of the agents will have to be continued, bad effects being met as they arise, and all possible adjuncts being used, including serums and blood transfusions. This conviction has grown out of an experience of complete failure before the use of sulfanilamide and its allies.

Sinus thrombosis can probably be dealt with successfully without the drugs if necessary. But here also the chemicals seem to hasten cure and prevent the danger of local and general spread of sepsis.

With petrositis, when one may not be sure that drainage and unblocking are sufficient, sulfanilamide might be withheld for further study of the case, unless there are signs of meningeal involvement or complete operative removal seems impracticable. I should try a drug before sacrificing the middle ear.

If one can consider labyrinthitis without entering into too much controversy, it seems proper, as Kopetzky has reported, to be more conservative, placing considerable reliance on the drug. Especially may one do so if one follows Alexander's dictum, to watch such a condition hour by hour and intervene only when there is definite sign of meningeal irritation. Recently 2 patients with acute diffuse suppurative labyrinthitis recovered with sulfanilamide, 1 without mastoidectomy.

Abscess of the brain should be treated surgically of course, but chemotherapy, unless it has produced severe toxicity, should be used to give additional protection against spread of infection or secondary meningitis.

DISCUSSION OF PAPERS IN SYMPOSIUM

DR. SAMUEL KOPETZKY, New York: I have always been in favor of transfusions and have used small ones repeated at two or three day intervals extensively. The minute one has mastered the sepsis in a particular case, the patient holds the hemoglobin after transfusion. I think sulfanilamide is the most valuable drug one has when one is faced with a grave situation. I do not think one should misuse it in the early stages. One sterilizes the blood and other fluids of the body, but the pressure on the nutrient veins of the intracellular walls of the

mastoid process is unaffected, and the lesion proceeds. Hence, this year many patients have come into the hospital with lesions such as epidural abscesses, which they should not have when treated by competent physicians, and they came because sulfanilamide was used in the early stages of the otitic infection and masked the progress of the pressure that produced the lesions. The misuses of sulfanilamide have not been sufficiently stressed, in my opinion. A patient receiving sulfanilamide should be kept out of direct sunlight. Most of the bad complications have been among Negroes in New York. Babies, also, must not be given large doses of sulfanilamide.

An easy means of holding the concentration is the rectal administration of sulfanilamide, which has not been mentioned. By means of a suppository put in the rectum one can hold the concentration steady for a longer time, and one can watch the result by testing for the concentration. At the least sign of discomfort, an enema removes it.

Sulfanilamide, plus transfusions, which replace materials used in the chemical combinations, leaves the body in better condition than ever. One should never stop the administration of the drug suddenly but should taper it off.

Dr. John Mackenzie Brown, Los Angeles: In conjunction with sulfanilamide my associates and I have been giving 50 to 100 cc. of antiscarlatinal serum every second day. There have been no ill results except slight urticaria.

Dr. William L. Culbert, New York: The first point I wish to make is the introduction of the duodenal tube into the stomach through the nose, for either occasional feedings or frequent small feedings, or even for feeding by the drop method used now in the treatment of gastric and duodenal ulcers. The second point is prolonged oxygen therapy, a powerful factor in the treatment of a toxic patient. The third is the use of leukocytic extract, which I have found of great value when the leukocytic battle was going against the patient.

Dr. John G. McLaurin, Dallas, Texas: My own experience is that few mastoidectomies are performed in my section, I believe because the physicians there generally are using sulfanilamide early. Many patients with acute infection of the upper respiratory tract or infection associated with otitis media have at first shown definite leukopenia. To my mind, it is indicative of a profound infection that has so affected the hemopoietic system that leukocytosis has not occurred. It shows lowered resistance. In such cases the use of sulfanilamide has almost invariably increased the leukocyte count. Another point brought out that I rather disagree with is that babies and young children do not tolerate the drug well. I find as a rule that a concentration of 4 to 5 mg. is sufficient to take care of the average infection of the upper part of the respiratory tract. Finally, one is given to believe that fever produced by sulfanilamide will usually disappear in forty-eight hours after administration of the drug has been discontinued. I have in some cases seen fever continue for two or three weeks.

Dr. Frederick M. Law, New York: After administration of sulfanilamide the roentgen appearance of the mastoid indicates that the condition is not as severe as the clinical evidence suggests. The drug does something to the contents of the cells whereby they are made more translucent to the roentgen ray, so much so that in cases in which the severity indicates operative intervention one cannot tell from a single film which side is involved. The opacity is practically gone.

The only change noted is in the appearance of the cellular structure. In the mastoid which begins to break down, the cells appear to be thin as compared with those on the other side. Stereoscopic films in the same case show the beginning of the osseous change; it may be faint, affecting a few cells only, or it may be extensive. Whenever one gives sulfanilamide one should notify the roentgenologist to that effect, and let him correct his interpretation. If a single film is made, the roentgenologist depends on the degree of opacity or on the appearance of a considerable area of coalescence. With stereoscopic films, one can see the

deeper cells; the ones in the floor of the mastoid, extending toward the antrum, are not there, or the cortical cells are present. On the single film one cannot see them.

It is apparent that if sulfanilamide is given before osseous change occurs resolution will take place. If it is given after osseous change has started the process continues, as subsequent films will indicate.

DR. THOMAS C. GALLOWAY, Evanston, Ill.: Many authors believe that sulfanilamide, to be effective, must produce definite results in from three to five days or, perhaps, seven. If no serious trouble develops, and if it appears to have some favorable effect, sulfanilamide should probably still be given. If then the clinical picture is not clear, and the condition still not serious, the use of the drug should be discontinued in order to show the true manifestations of the disease. Fever is most likely to complicate the picture. If the patient is otherwise doing well, it is usually safe to stop giving the drug and postpone the operation for two days to be sure the chemical is not responsible. Likewise, if an active disease seems to be cured, especially a deep process, it is generally advisable to give the drug from four to six days more, in the hospital of course, and to observe the patient closely thereafter for some time.

A SIMPLE QUANTITATIVE METHOD OF TESTING VESTIBULAR FUNCTION

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NEW YORK

Information regarding the functional activity of the eighth cranial nerve and its end organs is often required both for otologic and for neurologic diagnosis. In the case of the cochlear division this information is easily obtained, since the application of tuning fork tests is simple and their significance is generally known. Moreover, by means of the audiometer a graphic record can be obtained which is always available for comparison and which can be universally read and appreciated. With the vestibular division the case is very different. While all otologists are familiar with the principles of the Bárány tests of vestibular function, there is a remarkable lack of unanimity as to their value and the precise method of their application. Some rely chiefly on the turning test; others, on the cold caloric. In the performance of the latter, there are almost as many variations as there are testers. Out of such chaos accurate knowledge cannot come. What is urgently needed is a standard method which will serve as adequately and as simply for appraising vestibular function as Rinne's test does for appraising cochlear.

THE CONDITIONS

Such a test, to be of real value, must fulfil certain conditions.

1. It must be simple—applicable as well by junior interns as by heads of departments.

2. It must be time saving, not time consuming—Kobrak's quantitative method and others like it, admirable as they may be for the physiologic laboratory, are too cumbersome for the clinic.

3. It must test each vestibule separately—wherefore rotation, which stimulates both simultaneously though in different degrees, is unsatisfactory.

4. It must use a minimal adequate stimulus—one does not test the sense of touch with a hammer or of temperature with a red hot poker.

5. It must be quantitative—to allow of accurate comparison between ears.

6. Its results must be capable of simple expression so that they can be universally comprehended.

From the Department of Otolaryngology, New York Hospital and Cornell University Medical School.

THE METHOD

It is realized that it is impossible to devise a thermal test of absolute accuracy because the heat is conducted to the labyrinth by way of bone, and therein is considerable anatomic variation. Nevertheless, the cold caloric test performed in the manner to be described approximates as closely as possible to absolute accuracy, especially when it is remembered that what is being compared is always the two ears of the same patient, in whom anatomic characteristics are likely to be the same on both sides, and not two patients. In practice it has been found to be satisfactory, and it conforms with the requirements laid down.

The general principle of the method is that 1 cc. of ice-cold water is injected into the ear and the time of onset of nystagmus is noted. The ear is then emptied, and the time of duration of the nystagmus is also noted. These times bear a definite relation to each other under normal conditions, as will be explained later.

DETAILS

There are certain details which must be observed with some care if accurate and satisfactory results are to be obtained.

1. The position of the head is important. Ideally it should be in such a position that the external semicircular canal is in the *vertical* plane with the ampulla as the highest point. This is achieved as nearly as possible by leaning the head over to the horizontal so that one ear is above the other (the body has to lean over as well) and tilting the chin upward through 45 degrees by rotating the neck. The position sounds a forced and uncomfortable one, but actually it is not.[1]

2. The syringe used is of a special type. It has a setscrew on the plunger so arranged that only 1 cc. of the 10 cc. which the syringe contains when filled is delivered when the plunger is pressed down. By this means the inevitable change in temperature of the water in the syringe during transport from bowl to patient is minimized, the rise in temperature of the larger bulk of water (10 cc.) being infinitesimal, while that of 1 cc. only might be large.

3. The actual procedure is as follows: Ten cubic centimeters of water in which ice is floating—*ice* water, not *iced* water—is drawn into the syringe, which has lain immersed in the ice and water for some minutes. With the head in the position described so that the ear to be tested is uppermost, 1 cc. of the ice water is instilled into the external auditory canal, and the time of instillation is noted by stopwatch. The

1. Prof. Gusta Dohlman, of Lund University in Sweden, elaborated this position with me and suggested the large syringe.

patient is then made to direct the eyes on a finger held on the side opposite to the ear being tested, and the eyes are watched hawklike for the first flicker of nystagmus. This requires some practice to detect without use of artifice, but if the patient wears a pair of spectacles fitted with 20 D. lenses or the observer uses an ophthalmoscope, the onset of the nystagmus is more readily appreciated. This moment is noted on the watch. The interval which has elapsed between instillation and nystagmus is the *time of onset.*

At once the ear is emptied of water. The patient is tested for nystagmus, first with the head forward 30 degrees and then with it back 60 degrees to test all canals, and for pointing error, a matter of thirty seconds or so. He is then watched, again with great care, for the end point, when nystagmus stops; the time is taken again, and the second interval of time, from start of nystagmus to finish, is the *time of duration.* Finally the subjective reaction is noted, whether none, slight, moderate or severe.

The other ear is tested in the same manner after two or three minutes have elapsed, to insure the cessation of all dizziness.

4. If there is no reaction to stimulation in three minutes, the labyrinth is probably dead, but to make certain of this the test is repeated immediately for three more minutes. If still there is no reaction, the labyrinth can be pronounced dead with certainty.

5. Because patients are fearful of an unknown procedure they should be told before starting what is going to be done and that nothing more than a transient dizziness need be expected. It is also wise to test first that ear which is expected to be normal or the more nearly so, lest the other be hyperactive and the patient be disturbed and frightened by a severe reaction. By this method it is rare, however, for any serious discomfort to be felt, even with a hyperactive labyrinth, and vomiting is unknown.

CHARTING THE RESULTS

The results are represented as a simple fraction, the time of onset over the time of duration in seconds. A record is also made of the type and quality of the reactions obtained. The following example is from the case record of a patient with Ménière's disease.

		Right	Left
Nystagmus....	Time of Onset / Time of duration (sec.)	25 / 90	15 / 120
Reactions			
Nystagmus.....	Head forward 30°	To left	To right
	Head back 60°....	To left	To right (great discomfort)
Pointing error.....	.....	To right	To left (marked)
Subjective reaction	.....	Moderate	Severe with nausea

Opinion: Hypersensitive left labyrinth

READING THE RESULTS

It has been found that in the normal labyrinth tested in this way the average time of onset of nystagmus is about thirty seconds but that individual variations occur, so that responses varying from fifteen seconds as a minimum to sixty seconds as a maximum lie within the limits of normality.

More important than the actual time of onset, however, is the relation of this to the time of duration. Duration is normally about four times as long as onset. A very usual finding is 25/90, that for the right ear in the example given. If the time of onset is fifteen seconds, the low limit of normal, and that of duration no more than sixty seconds without undue subjective reaction, the reading can be considered within normal limits, but if with an onset of fifteen seconds the duration is prolonged much beyond sixty seconds, and pointing error is pronounced and subjective reaction considerable, as with the left ear in the example, the labyrinth is markedly hypersensitive. Similarly, if time of duration approximates to or is less than time of onset, particularly if the latter is sixty seconds or more, the function of the labyrinth is depressed. The relation of the two times, more than the times themselves, is the fact that matters.

For example: (1)

	Right	Left
Onset	60	60
Duration	200	200

or (2)

	Right	Left
	15	15
	60	60

indicates normal labyrinths. Though the responses are slow in (1) and fast in (2) they are in each case equal on the two sides and within normal limits.

But if the figures on the left side are reversed, thus:

(3)

	Right	Left
Onset	60	200
Duration	200	60

or (4)

	Right	Left
	15	60
	60	15

the reading indicates a markedly hypoactive left labyrinth. On the left side the relation of the times is wrong, and the reactions of the two sides are grossly unequal.

It is evident that by this means a factual report can be given of the function of the vestibule, a report which is as definite and accurate as an audiogram for the function of the cochlea. Moreover, a second observer, the neurologist for instance who receives the report from his otologist colleague, is not compelled to rely solely on the expressed opinion of the latter, but has before him the facts on which that opinion was based. Given a knowledge of the normal, which should be printed on each report sheet, he is in a position to check the other and form his own opinion. The argument applies with even greater force to published case reports.

Too often one finds such vague generalities as that "the labyrinth did not react to syringing with cold water," or reads with misgiving that with a hearing ear "the labyrinth was dead." Use of this method allows the observed facts to be given in small compass and facilitates the passage of accurate information. It would be of advantage to everybody if the cold caloric test could be standardized along these lines.

THE ROTATION TEST

But, it will be asked, what of the rotation test? Is that to be no more used? The answer is that compared with the cold caloric the rotation test is of little value. Rotation is a physiologic experiment, and as such has done yeoman service in helping to elucidate the problems of the labyrinth. As a clinical test of labyrinth function it fails because it stimulates both sides simultaneously, even if unequally, and because it is extremely difficult so to regulate the stimulus that it shall be minimal—the usual method produces a stimulus more nearly maximal. It still has a clinical purpose to serve, however, in differentiating between peripheral and central lesions. When the reactions to the cold caloric test are equivocal, the rotation test may help to elucidate their meaning, but the larger the experience with the cold caloric test the less frequently will rotation be found necessary.

CONCLUSION

The method which has been described of performing the cold caloric test has various advantages.

1. A known amount of water of known temperature, a temperature, moreover, easily achieved and easily kept constant, is allowed to act for the least time necessary to produce a reaction—thus it is quantitative. The rise of temperature which must take place in the water by contact with the body does not affect the quantitative character of the test, since the change is the same on the two sides, and it is two sides of the same patient which are being compared, not two patients.

2. The reaction is such that the end points are definite and their appearance can be timed—thus it is accurate, or at least as accurate as the eye of the observer, a proviso for human fallibility which has to be applied to all tests.

3. There is in normal ears a practically constant relation between time of onset and time of duration, 1 : 4—thus it is mathematical in the basis it provides for comparison.

4. The stimulus applied is minimal—thus the reaction of the patient is also minimal. Patients who have been tested by other and more

drastic methods are unanimous in their approval of this one and have no dread of it a second time.

5. The test is simple to perform, takes little time and fulfils all the conditions laid down at the beginning—thus it is practical. Its value in this respect and its accuracy have been amply proved by experience.

SUMMARY

The necessity for some standardization in the tests of vestibular function must be apparent to all clinicians who have to apply them and to all who have to peruse the written record of their results. The method described is simple, accurate, practical and easily recorded. Its value and accuracy have been attested by several clinics where it has been adopted as the routine method. It is suggested that its general adoption as the standard vestibular test would make for all-round simplicity.

123 East Sixty-First Street.

STRUCTURE OF THE PETROUS PORTION OF THE TEMPORAL BONE

WITH SPECIAL REFERENCE TO THE TISSUES IN THE FISSULAR REGION

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To the morphologist the petrous portion of the temporal bone presents several interesting features: It contains a bony case built in a unique manner to shelter the delicate terminals of the auditory and vestibular mechanism; this case is constructed from separate centers of ossification which ultimately blend so completely that there appears, at birth, a composite capsule with no line to indicate the originally separate entities. The capsule thus formed does not enlarge after birth, but increment is made in the petrous portion of the temporal bone in which the capsule is embedded. Even after adult size has been attained, histologic changes in the otic capsule continue throughout life; the site of the most striking postnatal alteration is the small area between the cochlea and the stapes, in the wall of the capsule; in this territory of the fissula ante fenestram the capsule retains nonosseous tissues (connective tissue and hyaline cartilage) to a degree not usually observed in adult "bones"; here also occur unusual forms of osseous tissue (intrachondrial and sclerotic bone).

To the histologist the organization of the petrous portion appears so different from that of any other bone in the body that he inevitably asks why in this region nature has transgressed the general rules governing the building of the bony skeleton; further, he is impelled to search for the basis of pathologic change in the structural peculiarities of the otic capsule.

An enlargement of certain features contained in a paper read at a meeting of the St. Louis Otolaryngological Society in December 1932.

From the Departments of Anatomy and Otolaryngology, Northwestern University Medical School. Contribution 293 from the former.

A summary of recent investigations into the structure of the temporal bone and of the ossicles which have been conducted under the auspices of the Central Bureau of Research of the American Otological Society; and, in part, a record of similar projects now under way at Northwestern University Medical School.

MATERIAL AND METHODS

The present report is based on the study of a large number of series in the otologic collection, from persons of various ages; the specific descriptions, however, are based on the following series, which are herein illustrated either as photomicrographs of sections or as photographs of wax plate reconstructions [1]:

Age	Series	Sections	Sections, Figures	Reconstructions, Figures
Fetus, term	4/18/30	219	7, 8	
		230	9	
		260	10	
Fetus, term	March '27			13
3 mo.	2/ 1/32	245	1, 2	
		250	3, 4	
		255	5, 6	
2 yrs.	10/26/32	...	.....	14
18 yrs.	1/17/31	...	.....	15
18 yrs.	2/26/30	...	.....	11, 12

OBSERVATIONS AND COMMENT

In any investigation into the formation and subsequent structural alteration of the temporal bone, it must be borne in mind that this skeletal element is, like all the others in the human body, impermanent. While it is recognized that the larger bones do not continue in the same state throughout life, this character of instability has only recently been studied in the temporal bone, the latter's otic capsule and the auditory ossicles.

Development of the temporal bone proceeds in a series of waves, affecting the several constituent parts at different stages. Within a few weeks, in the fetus, the otic capsule and the stapes attain approximately full size; subsequent growth is extracapsular and takes place in tissues which are destined to be mainly hemopoietic and pneumatic. But within the capsule itself, especially its fissular part, and within the stapes, gradual changes are going on which have to do with histologic metamorphosis rather than with growth; in the fissula certain of these changes are seemingly preparatory to the formation of otosclerotic bone.

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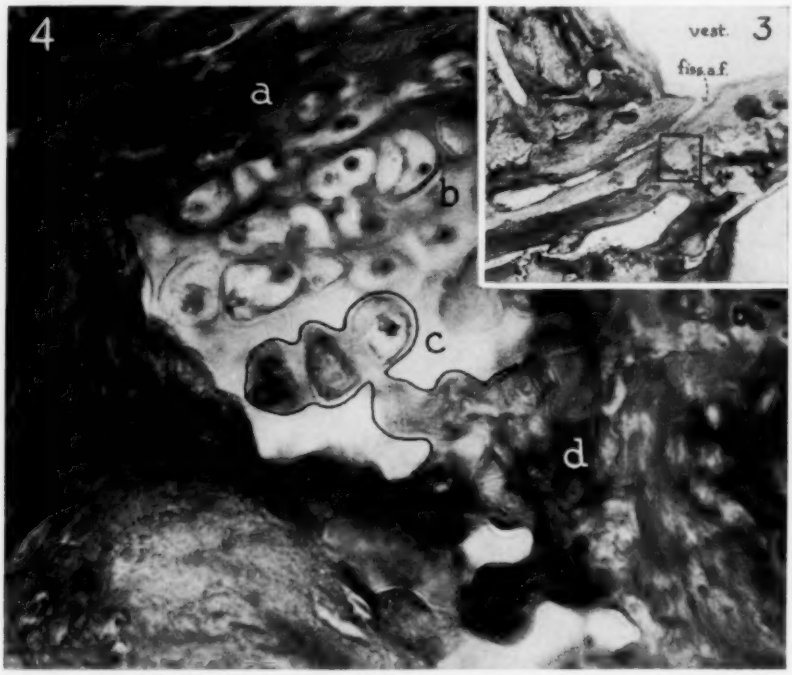
1. The photomicrographs were taken at the following magnifications: figures 1, 3 and 5 at 75 diameters; figures 2, 4 and 6 at 750; figures 7, 9 and 10 at 65, and figure 8 at 225. The reconstruction shown in figures 11 and 12 was prepared at a magnification of 50 diameters (by Dr. J. E. Karabin); a reconstruction of the space enclosed by the fissula in this specimen is illustrated by B. J. Anson and J. G. Wilson (The Fissula Ante Fenestram in an Adult Human Ear, Anat. Rec. **56**:383-393 [July] 1933 and Anson and Martin (1935) [11]; the reconstructions shown in figures 13 to 15 served as bases for drawings in articles by Anson and Martin (1935) [11] and Wilson (1935) [10]; they were prepared at a magnification of 125 diameters.

Figs. 1 and 2.—Photomicrographs of a horizontal section through the ear of an infant 3 months old. The area of bone is that part of the labyrinth bounded by the cochlea, the vestibule and the tympanic cavity. Figure 1 (× 40) represents a section through the otic capsule just anterior to the stapes and in the region of the vestibular end of the fissula ante fenestram, showing the abundance of intrachondral bone and the relation of such bone to the marrow spaces (see especially the small area enclosed by lines). Figure 2 (× 600) represents an enlargement of the area marked out by lines in the preceding figure, showing a finger-like process of the resistant intrachondrial bone projecting into the loose and vascular connective tissue of a marrow space. The margin of the intrachondrial bone is indicated by a thin line.

In these and the succeeding figures, *ant.* indicates the anterior aspect; *b.st.*, the base of the stapes; *car. canal*, the carotid canal; *cart.*, cartilage; *cochl.*, the cochlea; *c.t.* or *conn. tissue*, connective tissue; *f.e.f.a.f.*, the fenestral extremity of the fissula ante fenestram; *fiss.a.f.*, the fissula ante fenestram; *int. ac. meatus* or *int. acoust. meatus*, the internal acoustic meatus; *lat.*, the lateral aspect; *lat. semic. canal*, the lateral semicircular canal; *med.*, the medial aspect; *mm.*, mucous membrane; *modiol.*, the modiolus; *post.*, the posterior aspect; *spir. lam.*, the spiral lamina; *spir. tract*, the spiral tract; *st.l.*, the stapedial ligament; *t.c.*, *cav.t.* or *tymp. cav.*, the tympanic cavity; *t.e.* or *t.e.f.a.f.*, the tympanic extremity of the fissula ante fenestram; *v.*, *vest.* or *vestib.*, the vestibule; *v.e.* or *v.e.f.a.f.*, the vestibular extremity of the fissula ante fenestram, and *vest. window* or *vestib. window*, the vestibular window.

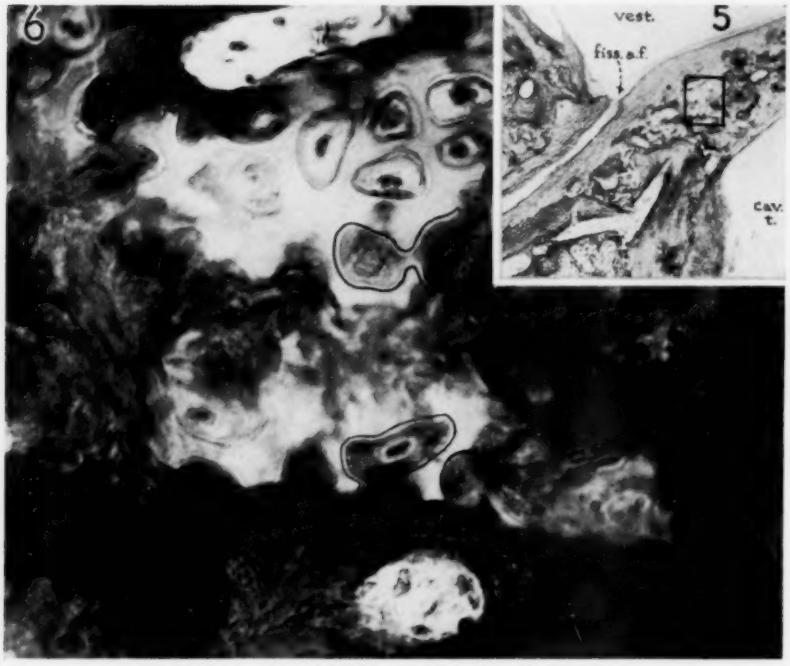
1. *Stapes and Vestibular Window.*—In middle intrauterine life the stapes and the window in the otic capsule which receives the stapedial base are as large as they will ever be; but, although full size has been reached at this early stage, internal alteration, resulting in modification of external form, continues throughout life.

The base of the stapes attains "adult" dimensions in the embryo of 161 mm.; at this stage the first center of ossification has appeared on the tympanic surface of the base; thereafter, while the latter surface becomes fenestrated and the entire base hollowed, the vestibular surface remains covered with a layer of hyaline



Figs. 3 and 4.—Photomicrographs, continued, from a succeeding section in the same series. Figure 3 (× 40) represents a section through the otic capsule in the region of the fissula, showing the typical hyaline cartilage (surrounding the end of the fissula), the modified cartilage of the intrachondrial bone and the endochondral bone. Figure 4 (× 600) represents an enlargement of the area marked in the preceding figure (situated on the lateral wall of the fissula ante fenestram, near the vestibular extremity). At the top of the figure, from the surface inward, are observed transitional changes in the alteration of the cartilage cells and the containing lacunas (at *a* and *b*); toward the bottom are seen further stages in the conversion of lacunas into osseous capsules and the manner in which these become attached, in pedunculated form (at *c*), to the surrounding bony wall (at *d*). A stalk with a terminal cluster of cells in lacunas is outlined (at *c*).

cartilage (Anson, Karabin and Martin, 1938 [2] and 1939 [3]). Between the 183 mm. stage and term, the fenestrated internal wall disappears, and the base is reduced to a relatively flat plate, cartilaginous on the vestibular, and osseous on the tympanic, surface. Although it continues throughout life as a bilaminar plate, cartilage gradually gives way to bone. In infantile and juvenile stages the base of the stapes is relatively thick, cartilage covering the vestibular aspect completely and also occurring on the tympanic surface in the form of small patches. In adult stages, however, ossification has advanced to a point at which bone presses through the cartilaginous lamina, to appear on its free surface as islets; in the adult of 18 these are small and isolated, and at 57 some have coalesced to produce larger areas of bone, while in the subject of 70 bone covers approximately half of the surface. Concurrently, the entire base becomes thinner, both laminas alike being affected.



Figs. 5 and 6.—Photomicrographs, continued, from a succeeding section in the same series. Figure 5 (× 40) represents a section through the region of the fissula and the inferior aspect of the vestibular window, showing the arrangement of cartilage, intrachondrial bone and lamellar bone on the tympanic wall of the vestibule. Figure 6 (× 600) represents an enlargement of the area marked in the preceding figure. The series of changes noted in figure 4 are here again apparent. At the right of the field a modified, ossifying capsule is attached to the bony wall of the cartilage island by a thin pedicle; in the lower middle, one more thickly encapsulated is attached by a broader connection (each outlined). Others have become enclosed in the bone and appear as marginal crenations in a marrow space.

2. Anson, B. J.; Karabin, J. E., and Martin, J.: Stapes, Fissula Ante Fenestram and Associated Structures in Man: I. From the Embryo of Seven Weeks to That of Twenty-One Weeks, Arch. Otolaryng. **28**:676-697 (Nov.) 1938.

(Footnotes continued on next page)

The crura also attain their full length in the 161 mm. embryo, but are still solid cartilaginous bars; replacement of cartilage by bone progresses so rapidly that in the 183 mm. embryo the crura are hollow osseous cylinders, perforated by vessels along their internal surface. Further resorption converts these hollow cylindric members into channeled structures, with their troughlike excavations mutually facing each other. Reduction in bulk, however, does not terminate with the attainment of sulcate form, since they become somewhat thinner as age advances.

The neck and head of the stapes are still continuous parts of a solid cartilaginous cylinder in the embryo of 183 mm., at which stage the crura have become hollow columns composed entirely of bone; but in the fetus at term, cartilage remains only on the articular surface of the head, the capital and cervical portions otherwise being osseous. In the adult a slow process of excavation alters the form of the neck and the head, often resulting in the removal of their superior surfaces.

Keeping pace with the changes which, in their steady advance, so completely modify the parts of the stapes, the window which receives the ossicle undergoes a similar morphogenesis. The window in early embryos is irregularly triangular, the base of the stapes being relatively much smaller than the fenestral space; in the embryo of 161 mm. it has reached adult dimensions but not the typical adult form. The window is lined by a rim of cartilage throughout life; this fenestral cartilage diminishes as age advances, in old persons being approximately one half as thick as it is in midfetal life; yet it is never wholly replaced by bone. Representing a persistent portion of the primordial otic capsule, the cartilaginous fenestral rim possesses an extension at each extremity; the anteroposterior prolongation contains the fissula ante fenestram; the posteroinferior one surrounds the fossula post fenestram.[4]

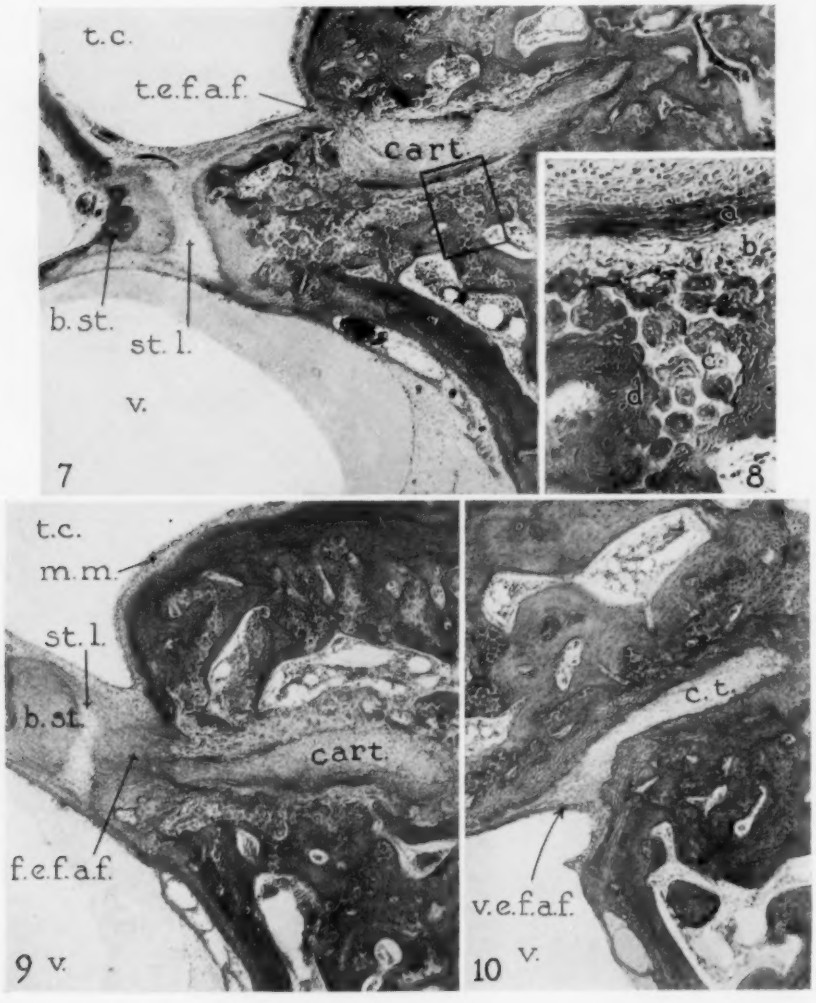
2. *Semicircular Canals.*—In the embryo of 8 weeks the semicircular canals are still encased in cartilage; since their size will have increased by five times when maximum dimensions have been attained, "the tremendous growth and expansion of the canals must take place within this firm tissue" (Bast, 1932[5]). This expansion is accomplished

3. Anson, B. J.; Karabin, J. E., and Martin, J.: Stapes, Fissula Ante Fenestram and Associated Structures in Man: II. From the Fetus at Term to the Adult of Seventy Years, Arch. Otolaryng. **29**:939-973 (June) 1939.

4. The stapes, vestibular window and neighboring structures are now being studied in later fetal stages (3 month to term), by means of series which have been lent by Professor Bast. It may be recorded here, after a preliminary study of these fetal series, that in the specimen of 147 mm. crown-rump length the stapes is still entirely cartilaginous and that calcification of the cartilage, with concurrent deposition of periosteal bone, is under way in the base of the stapes from a fetus of 150 mm., while in specimens of 160 and 163 mm. excavation of the cartilage is already a striking feature. At 205 mm., the fenestrated bone on the facing surfaces of the crura and on the tympanic surface of the base has been largely removed, and new bone covers part of the cartilaginous lamina; the base is completely bilaminar in the fetus of 260 mm., at which stage its general structure and form are similar to those of the fetus at term. During the course of these changes the stapes retains the same over-all dimensions; when once bone appears, growth ceases.

5. Bast, T. H.: Development of the Otic Capsule: I. Resorption of the Cartilage in the Canal Portion of the Otic Capsule in Human Fetuses and Its Relation to the Growth of the Semicircular Canals, Arch. Otolaryng. **16**:19-38 (July) 1932.

through a wide destruction of the cartilage situated between the arcs of the canals, a localized dedifferentiation of cartilage at the advancing margin of each canal with concurrent rebuilding of cartilage at the



Figs. 7 to 10.—Photomicrographs; horizontal sections through the fissular area in the ear of a fetus at term. Figure 7 (× 39) represents a section through the tympanic extremity of the fissula ante fenestram; cartilage has replaced the earlier connective tissue of this lateral fissular orifice. Figure 8 (× 135) represents an enlargement of the area indicated in the preceding figure, showing the histologic succession of tissue at the margin of the cartilage mass. The letters indicate stages substantially as in figure 4. Figure 9 (× 39) represents a section through the auxiliary, or fenestral, orifice of the fissula; here, likewise, cartilage occupies the opening. Figure 10 (× 39) represents a section through the vestibular extremity of the fissula; connective tissue remains at this medial orifice.

opposite receding margins. Approximately maximum growth, according to Bast, is reached in the midfetal stage, when ossification centers make their first appearance in the otic capsule. This means that, like the stapes and the vestibular window, the canalicular portion of the capsule is as large as it will ever be at the time the fetus is entering the second half of its period of intrauterine development.[6]

3. *Cochlea.*—The cochlea, too, is approximately as large in the fetus of 4½ months as it will be in the adult and therefore belongs in a category with the portions of the ear already described. A slight enlargement may occur through expansion of the shell into the marrow space which regularly surrounds the cochlea in the late fetus and which still persists in the ears of some infants and children. This space matches roughly the form of the cochlea. Its outer wall is endochondral bone; its inner is hyaline cartilage and intrachondrial bone (see hereinafter). The latter tissues and the space which bounds them externally are reduced as age advances—perhaps partly by expansion of the cochlea through rebuilding of its wall.[7] But since such growth, if it occurs at all, must be moderate, it may be said of the entire otic capsule that ossification is a process of differentiation unaccompanied by enlargement; unlike skeletal elements generally, it does not broaden or lengthen during the stage in which its cartilage is being replaced by bone.

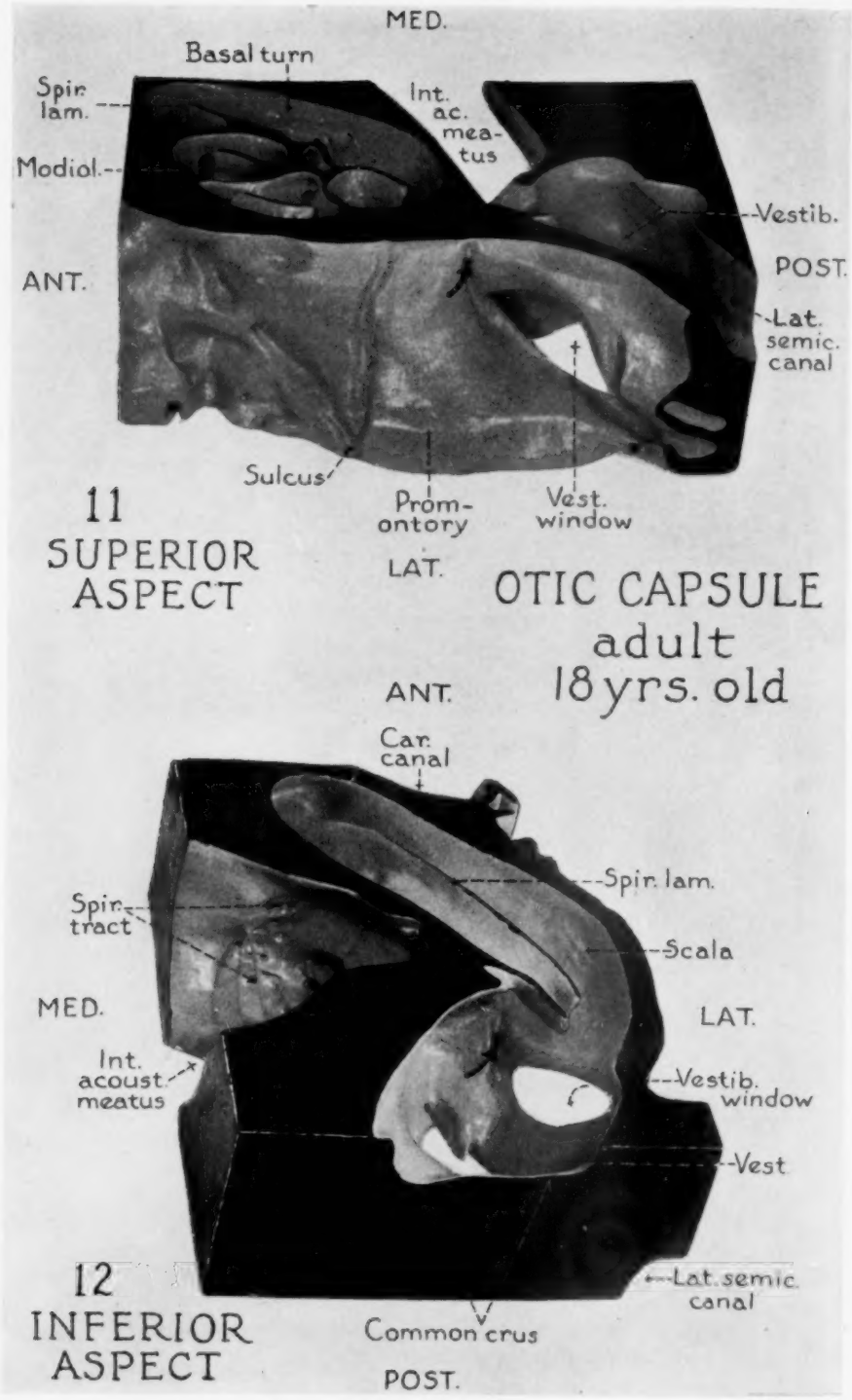
4. *Fissula ante Fenestram.*—In the area of the otic capsule just in front of the vestibular window, between the latter and the cochlea, is situated a stripe of connective tissue; continuous externally with the submucosal connective tissue and internally with the perichondrium or periosteum of the vestibule, this fibrous seam in the bone occupies a channel termed the fissula ante fenestram. Like the capsule, of which it is a constituent part, the fissula has acquired adult dimensions when the fetus has but reached the halfway mark in its intrauterine life.[8]

Typically in specimens of temporal bone from adults, the fissular channel begins in a small opening on the medial tympanic wall just anterior to the vestibular window (fig. 11, at arrow); extending at first inward toward the cochlea, it next turns backward upon itself,

6. The early attainment of full growth by the capsule and the ossicles is a striking phenomenon when it is considered that the weight of the human body will increase by fourteen times between the middle of fetal life and birth and by approximately three hundred times between the former stage and that of the 18 year old adult.

7. A study of the changing character of the tissues which form the immediate wall of the cochlea is in progress. It is hoped that, through the preparation of reconstructions of the constituent tissues, something may be learned about the function of intrachondrial bone.

8. In point of fact, owing to individual variation, the channel may actually be larger in the fetus than in some adults (Anson, Karabin and Martin, 1939,[3] figs. 60 to 64).



Figs. 11 and 12.—Reconstruction of the otic capsule; fissular orifices at arrows; ×7½.

descending as it does so; it ends on the anterior wall of the vestibule at or near the point of continuity of the latter with the scala vestibuli of the cochlea (fig. 12, at arrow; also, figs. 1, 3 and 5). Variation in form and size is common: It may be short and slitlike or long and tortuous; it may be thin or bulbous.[9]

But more important than variation in shape of the fissula is variation in contents (see the paragraph headed "Primary Cartilage"). The area is commonly found to be in a state of histologic flux (Wilson, 1935 [10]). The most striking result of cellular disequilibrium is the production of unusual tissues, for example the osteoporotic type of bone present in cases of otosclerosis (Anson and Martin, 1935 [11]).

The bone enclosing the fissula often measures not more than 0.5 mm. in width, 1.2 mm. in length and 1.6 mm. in height, but in spite of the small size, great interest attaches to its histologic fate, since it is here that sclerotic bone occurs with such frequency that the region has come to be known as the "site of predilection" for otosclerosis. Small as is the fissula, the vestibular window is but little larger; consequently, any deforming effect which changes, initially fissular, may have on the neighboring window, will be vitally important: Newly formed bone, by encroaching on the space, will reduce the excursion of the stapes and thereby cause a loss of acuity in hearing. Although it is recognized that these alterations are not "pathologic" in the sense in which the term is employed in the description of, say, osteosarcoma, they nevertheless produce results which certainly are abnormal. In order to appreciate these effects fully, it is necessary to think in terms of slow progress, unlike the dramatic advance which marks the spread of neoplasms, and to recognize that alterations in structure which would be infinitesimal in the gross pathologic picture of other members are here of grave importance.

The region of the fissula ante fenestram is, then, one of vital importance, since spongious foci, appearing originally in relation to the fissula, may spread into the fenestral space and so involve the base of the stapes. In order to understand the principles of fissular morphology, it is necessary first to review the stages in the development of the capsular bone and in the formation of the special group of tissues in the territory of the fissula.

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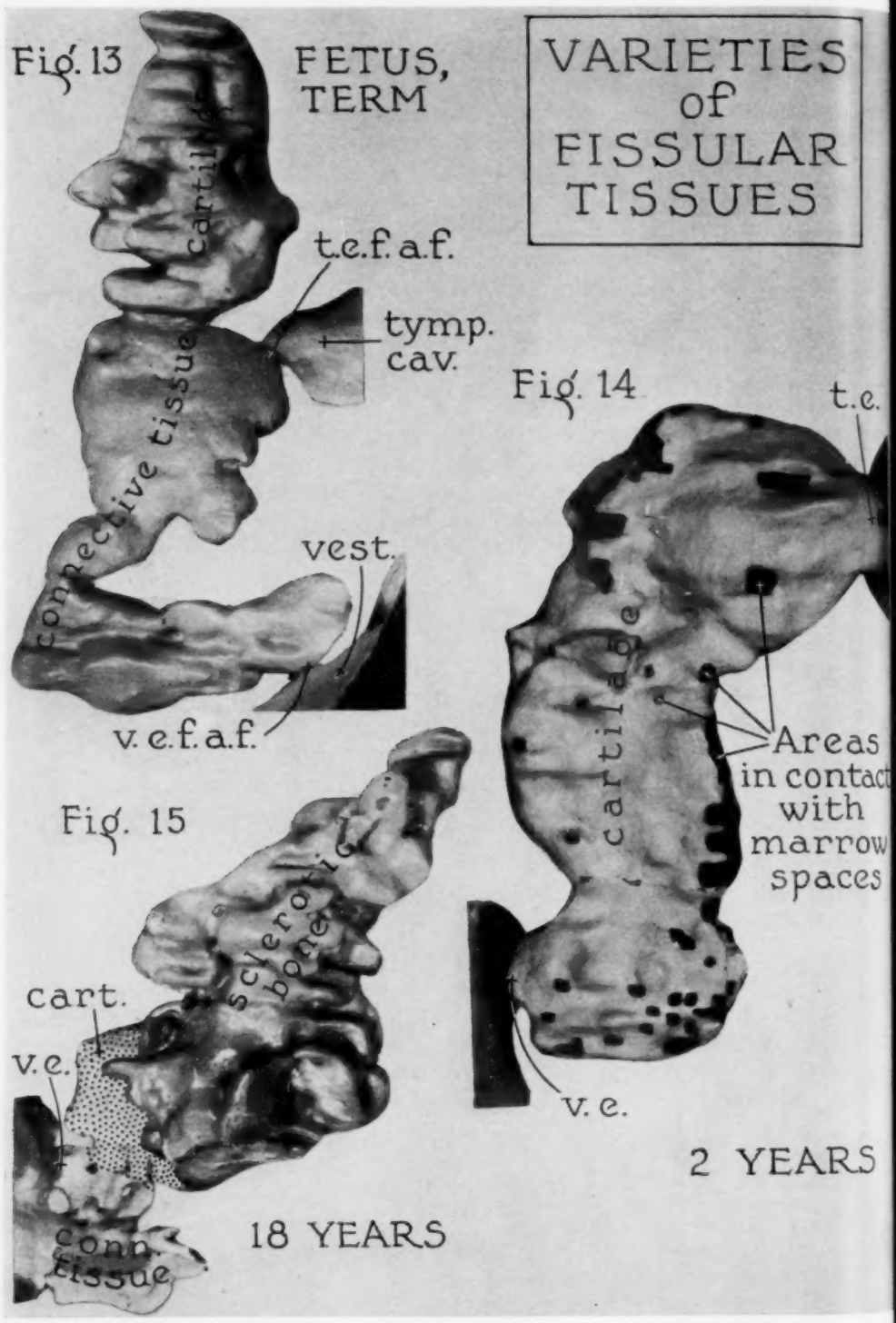
9. Selected specimens, exhibiting variety in form, terminations and contents have been described by Bast and by Wilson, Anson and associates in articles which will be referred to hereinafter. The fissula is not infrequently incomplete, lacking one or both orifices. The extremities of the fissula may open, not near the vestibular window but into the semicanal for the tensor tympani muscle; the vestibular orifice may open, not into the vestibule proper but into the scala.

10. Wilson, J. G.: Fissula Ante Fenestram and the Adjacent Tissue in the Human Otic Capsule, Acta oto-laryng. **22**:382-389, 1935.

11. Anson, B. J., and Martin, J.: Fissula Ante Fenestram: Its Form and Contents in Early Life, Arch. Otolaryng. **21**:303-323 (March) 1935.



Figs. 13 to 15.—Reconstructions of fissular contents; × 42. Figure 13, a medial view of the reconstruction, shows connective tissue and cartilage. Figure 14, an anterior view, shows cartilage. Figure 15, a medial view, shows cartilage (stippled) and sclerotic bone.

The cartilaginous otic capsule of the primitive chondrocranium is converted into an osseous case through the production of bone from a number of centers of ossification.

Within each of the centers of ossification, as shown by Bast (1930),[12] a succession of changes occurs, some of which match those which take place in any long bone in the human body, while others are peculiar to the otic capsule.

Periosteal (or perichondrial) bone appears first, in the form of a layer investing the primordial cartilage; its matrix is deposited by cells derived from the perichondrium of the otic capsule; it closely resembles in every respect the comparable tissue of any developing long bone.

The enveloping plate thus constructed is foraminous, permitting the entrance into the subjacent cartilage of buds of vascular tissue, whose source is, again, the perichondrium. Individual cells from the osteogenetic buds invade lacunas of the necrotic cartilage and excavate the latter tissue. Then, as in membrane bone generally, endochondral (or primary) bone is produced in the excavated spaces. Usually it remains in the capsule throughout life, little of it being replaced by haversian (or secondary) bone.

A third and special type of bone is formed in the capsule, as a result of the rapidity of invasion. When the osteogenetic buds become vascular, their growth is exceedingly rapid; sufficient time, apparently, is not allowed for complete removal of the cartilage matrix. As a result, areas of hyaline cartilage are left standing between the invasive buds. The lacunas of the partially destroyed cartilage are invaded by the cells of the osteogenetic buds; these, becoming osteoblastic, deposit bone around themselves on the wall of the lacunas, giving to the tissue the appearance of a calcified cartilage. These cartilage islands (interglobular spaces, or "intrachondrial bone" of Professor Bast's descriptions) are to some extent destroyed; osteoclasts excavate a channel through the calcified wall, removing the matrix and contained cells and replacing the calcified cartilage with endochondral bone. Since intrachondrial bone is, generally, not as widely distributed in the old as in the young, it seems safe to assume that this process continues in postnatal stages.

The process of ossification of the otic capsule begins in about the sixteenth week of intrauterine life. Centers appear in rapid succession and fuse to produce a complete osseous capsule in the fetus of approximately 22 weeks (Bast, 1930[12]). The fissular region, however, is slow to ossify, appearing as a considerable mass of cartilage in the fetus of 21 weeks (183 mm.); it extends from the cartilaginous rim of the vestibular window to the cochlea, forming part of the latter's posterolateral wall (Bast, 1930,[12] figs. 25 and 27; Anson, Karabin and Martin, 1938,[2] figs. 20 and 21). At the core of this seam of cartilage is a strip of connective tissue which extends through the capsular wall from the tympanic to the vestibular surface; on the outer aspect of the cartilage is an investment of periosteal bone from which project trabeculate formations of endochondral and intrachondrial bone. The osseous shell,

12. Bast, T. H.: Ossification of the Otic Capsule in Human Fetuses. Publication 121, Carnegie Institution of Washington, 1930; Contrib. Embryol. **21**:53-82 (June) 1930.

by accretion, becomes part of the general petrous mass; the cartilage is seemingly reduced in bulk by slow conversion into "cartilage islands."

Established, then, at the midfetal stage is the association (in the small area just in front of the vestibule) of fibrous, chondral and osseous tissues: Connective tissue is embedded in cartilage; the latter is encased in bone (figs. 1, 3, 5 and 10).

These tissues, initially present, and others, which in some specimens replace them, will now be discussed.

(*a*) Connective Tissue: Connective tissue is the usual occupant of the fissular channel. It is formed by a process of dedifferentiation of the cartilage composing the primordial capsule (Bast, 1930[12]); the process is similar to that which occurs in the expanding canalicular area, as has been described. Whether examined in the late fetus or in the old adult, it is found to be but slightly vascular. At its margins the fibrous tissue passes gradually into cartilage or bone, through a perichondrium or a periosteum as an intermediary tissue.

(*b*) Primary Cartilage: That part of the original capsular tissue in which the connective tissue is embedded persists as a more or less complete chondral tube throughout life (figs. 1 to 6). Large in the 183 mm. fetus (Anson, Karabin and Martin, 1938,[2] figs. 20 and 21), it regularly decreases in thickness as age advances. The cartilage is apparently taken over into endochondral or intrachondrial bone by slowly progressing osteogenesis, since in some old subjects it remains as a readily distinguishable layer only at the vestibular extremity of the fissula, still continuous, however, with the fenestral cartilage (Anson, Karabin and Martin, 1938,[2] figs. 22 and 23); through the body of the fissula and at the latter's tympanic orifice in such instances the fibrous tissue rests directly against the osseous tissue of fissular wall.

(*c*) Calcified Cartilage (Cartilage Islands): Part of the wall of the fissula in late fetal and in postnatal stages is composed of intrachondrial bone. Where the primordial cartilage persists as a tube external to the connective tissue and internal to the bone, the intrachondrial tissue—midway in structure between cartilage and true bone—exists as a stratum of varying thickness on the outer aspect of the cartilaginous tube. As has been explained this intermediate type of tissue is formed through the ossification of remnants of cartilage which were left undestroyed by the invasive osteogenetic buds; thus sequestered during the period of fetal development, it endures, as a histologic half-caste, in the otic capsules of persons 70 and 80 years of age.[13]

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13. The histologic mechanism by which a cartilaginous matrix is converted into a "bone within cartilage" has been fully described by Bast (1930).[12] The persistent hyaline cartilage is gradually converted into a peculiar sort of calcified tissue; osteogenetic buds, derived from the perichondrium, invade the lacunas, to replace the cartilage cells and deposit a bony shell around themselves on the inner aspect of the original lacunas.

In the region between the cochlea and the vestibular window (i. e., the fissular region), this hybrid tissue is especially abundant, but from a site in the fissular wall it may extend deeply into the surrounding capsule (figs. 3 to 8). In passing from the chondral lining of the fissular space into the deeper tissue, a gradual histologic transition is encountered, beginning with cartilage and ending in true bone, the intermediate tissue being the "cartilage islands." In the layers bordering the fissular channel the cartilage cells lie in typical lacunas, composed of hyaline material homogeneous in texture. Nearer the bone the lacunas are larger and the intracellular material more deeply stained. Where cells are actually related to bone (as at the margin in fig. 4) they are encapsulated by a tissue which is definitely osseous. Various degrees of inclusion are frequently seen in the same section (figs. 5 and 6); attachment may consist of a narrow pedicle or of a broad stalk; finally the cell may be buried in bone, so that, with its neighbors, it lends an appearance of crenation to the periphery of the cartilaginous area. The cartilage is avascular; wherever vessels occur they are surrounded by bone, although this osseous rim is in some instances exceptionally thin.

Once formed, the intrachondrial bone is apparently a more resistant tissue than the endochondral bone; frequently it is found projecting prominently into the marrow spaces as tonguelike or finger-like prolongations, continuous with collections of the same tissue which are embedded in the ordinary bone of the capsule (figs. 1 and 2). The jagged form of these projections indicates that some process of halisteresis has been operative, but less successfully against the intrachondrial than against the endochondral bone.

These persistent remnants of cartilage—changed from their original form without loss of histologic identity—vary in abundance in different persons of the same age and in persons of different ages. Usually, they are more widely distributed in late fetuses and in children than they are in old adults; in the ears of both the young and the aged they are likely to be most plentiful in the innermost layers of the otic capsule, lying adjacent to the labyrinthine wall; the "islands" are often concentrated in that portion of the wall which, intervening between the cochlea and the vestibular window, houses the fissula ante fenestram.[14]

14. This particular phase of capsular metamorphosis is being studied in a large number of series representing ages between that of the late fetus and that of the adult of advanced years. It may be stated now, however, that the differences noted in the abundance of the intrachondrial bone are probably established during the ossification of the otic capsule. As Professor Bast has pointed out, in recent discussion of our otologic studies, the destruction of cartilage is exceedingly rapid in the otic capsules of some fetuses and slow in those of others; when it is rapid, few islands of cartilage remain to serve as a framework for intrachondrial bone; when it is slow, such patches persist to behave as nuclear centers for the deposition of bone.

When, as has been previously mentioned, the cartilaginous lining of the fissular channel is poorly represented, intrachondral bone is likewise sparingly distributed in the immediate wall of the fissula. Whether replaced through a tardy process of osseous encroachment or sparsely present from the fetal period onward, the intrachondrial tissue is still usually to be found at the vestibular extremity of the fissula ante fenestram. In such cases, which ordinarily are those of old adults, the fibrous content of the fissula rests against the bone except at the medial fissular orifice, where cartilage intervenes.

(*d*) Endochondral Bone: The true bone of the fissular region is primary bone, like that which makes up the bulk of the entire capsule.

As Bast has shown, ossification of the otic capsule is progressing rapidly in the 161 mm. stage; in the fissular region it begins on the medial tympanic wall and extends into the cartilage toward the fissular tract of connective tissue (Bast, 1933,[15] figs. 9 and 10). In the fetus of 210 mm. periosteal (perichondrial) bone has spread to the walls of the vestibule and the cochlea and along the course of the fissula (Bast, 1933,[15] figs. 13 to 15). In the 305 mm. fetus endochondral bone is beginning to obliterate the primitive marrow space bounded by the tympanic, vestibular and cochlear plates of perichondrial bone (Bast, 1933,[15] figs. 21 to 24). Part of this endochondral bone is deposited on the intrachondrial tissue; the latter then remains in the form of islands when endochondral bone has largely displaced marrow.

Modification of the bone next ensues, and it soon becomes impossible to distinguish the two types, which during the period of fetal ossification were characteristic in position and configuration.

In respect to density, the capsular bone, in postnatal stages, exhibits striking variations; in some specimens the bone is "petrous," containing few marrow spaces, while in others marrow spaces are large and numerous and the intervening trabeculae small. Occasionally, the marrow occupies large cavities continuous with the space of the fissula ante fenestram (Anson and Martin, 1935,[11] fig. 58; Wilson, 1935,[10] fig. 10). It is conceivable that within the otic capsule marrow spaces increase with advancing age, to produce a condition of rarefaction comparable with that occurring in the stapes.

(*e*) Secondary Cartilage: The fissular tube of primary cartilaginous origin is relatively bulky at the midfetal stage; as already described, it extends anteriorly to the cochlea and forms part of the latter's wall. In later stages this cochlear extension is replaced by bone and the fissular cartilage reduced to surround a fibrous core.

While the original cartilage which forms the wall of the fissular channel is thus progressively encroached on, new cartilage, in the form of a solid mass, may come to occupy all or part of the channel, replacing

15. Bast, T. H.: Development of the Otic Capsule: II. The Origin, Development and Significance of the Fissula Ante Fenestram and Its Relation to Otosclerotic Foci, Arch. Otolaryng. **18**:1-20 (July) 1933.

the original connective tissue. Such replacement occurs commonly in the late fetus, the infant and the child. In a recent examination of twenty-seven series of temporal bones from fetuses at term, infants and children (to the age of 10 years) the fissular tract in 16 was found to be occupied by a cartilaginous mass. The form of such chondral masses has been described and figured by Anson and Martin (1935),[11] Wilson (1935)[10] and Bast (1936).[16]

As shown by Bast (1936),[16] the cartilaginous masses of secondary formation appear for the first time in older fetuses; the earliest stage in which one was noted was a fetus of 180 mm. (21 weeks). The manner of its origin and spread and the mechanism by which it is subsequently reduced in bulk or replaced by a more active tissue will now be considered.

The new cartilage, which, as a secondary growth, comes to occupy a fissular channel once filled with fibrous tissue, is derived from the dormant shell of similar tissue which lines the channel (Anson and Martin, 1935[11]; Bast, 1936[16]), there being a gradual histologic transition from perichondrial connective tissue to newly formed cartilage.[17] Bast (1936)[16] stated that this replacement is an attempt on the part of the bone to reduce the extent of the fissular space, since the activated cartilage appears at an embryologic age when ossification of the capsular cartilage should have been completed and later may undergo ossification marginally.

In the temporal bone from a child 2 years old the occurrence of precociously developed cartilage within the fissular space makes clear the manner in which the new tissue gradually displaces the original connective tissue of the fissula to produce a chondral nodule within the bone between the cochlea and the vestibular window. At the tympanic orifice the new cartilage bulges into the semicanal of the tensor tympanic muscle; at the vestibular extremity it pushes toward the mouth of the orifice, where some of the original connective tissue still remains; at intermediate points it presses into marrow spaces (fig. 14, dark areas). At each extremity of the mass there is a gradual transition from perichondrial connective tissue into newly formed cartilage.

Of a different order, of course, is the cartilage which lines the fissular orifice and against which the newly formed cartilage lies; it is mature tissue which has been present in this locality from the stage

16. Bast, T. H.: Development of the Otic Capsule: III. Fetal and Infantile Changes in the Fissular Region and Their Probable Relationship to the Formation of Otosclerotic Foci, Arch. Otolaryng. **23**:509-525 (May) 1936.

17. It should have become clear to the reader that the fissula in histologic preparations is never a true fissure. Presumably because the fissula ante fenestram was first studied in dried bone by the simple method of probing, the structure is spoken of as if it were a true space (see bibliography in Bast, 1930[12]).

of embryonic formation. It passes imperceptibly into the intrachondrial bone which intervenes between the mass of young cartilage and the bone (figs. 7 and 8); at no point does the mature cartilage of the fissular shell or the intrachondrial bone pass by an intermediate transitional tissue into the young (activated) cartilage which occupies the fissula. It may, therefore, be said that the cartilage in many young specimens occurs not only as a parietal investment for the connective tissue of the fissula but as a nodular mass also, which partially or wholly replaces that itssue.

Occasionally the nodule is situated above the level of the tympanic orifice of the fissula. In a fetus at term, for example, the lower portion of the fissula terminates above and below in orifices that are occupied by connective tissue (fig. 13), but in an upward extension of the channel, two thirds as long as the fissula proper, the activated cartilage occurs as a discrete mass. Assumedly, in further spread, the cartilage would have taken the place of the connective tissue from the tympanic to the vestibular extremities.

The cartilage mass is related, in some specimens, not only to the tympanic extremity (fig. 7) but to an auxiliary fenestral extremity (fig. 9) where it is mergent with the lining of the vestibular window; as in the specimen illustrated, it frequently fails to reach the vestibular orifice (fig. 10). Here again, the new cartilage is set off sharply from the fissular shell, the tissue of the latter, however, passing by gradual transition through intrachondrial bone into the endochondral bone of the capsule (fig. 8)—wall and content remaining separate entities.

The tissue of the chondral nodule, having replaced the fibrous portion of the fissula, undergoes alteration within its own mass. Once formed, its matrix changes character, staining deeply with hematoxylin; since this is what happens in cartilage generally just prior to invasion by osteogenetic buds, it appears that the cartilage "has reached the final stage preceding the process of ossification." Supportive evidence is seen "in some of the older ears, in which the cartilage mass is being replaced by osteoid tissue or even by intrachondrial bone" (Bast, 1936[16]). Through the operation of such a process the occurrence of isolated masses of cartilage is explained (Anson and Martin, 1935,[11] figs. 46 to 49); in such cases the channel is incomplete, the expected site occupied by endochondral bone and discontinuous chondral nodules.[18]

18. It is present opinion that the fissula is not as frequently obstructed in the old as in the young—a baffling circumstance, if true. But it is not definitely established that the regular content of the adult fissular channel is connective tissue. The fissula is now being studied in a large series of sections, and in the following three groups: fetuses at term to children of 10 to 12 years, older children to young adults (21 years) and adults to the age of 80. A comparison of types by ages should put light on the problem of the ultimate fate of the fissular tissues.

Except for local protrusion of the cartilage into marrow spaces opening on the fissular wall (fig. 14), there is no indication that the activated tissue invades the surrounding capsule; so far as observed, it never occasions atrophy of the fissular shell.

(*f*) Sclerotic Bone: When sclerotic bone occurs within the confines of the fissula, however, the primary bone of the otic capsule may be replaced to some extent, since this new osseous tissue is mildly invasive; spreading, it may replace the quiescent cartilage of the vestibular window and appear in close proximity to the base of the stapes (Wilson and Anson, 1933,[19] plates I and IV, Bast, 1933,[15] fig. 36).

In a specimen from an adult 18 years old the fissular area is remarkable for the manner in which it is occupied by a lump of sclerotic bone (fig. 15). At the inferior fissular extremity fibrous tissue gives way to cartilage, the latter then merging with vascular, spongy bone of the sclerotic type, which extends anterosuperiorly toward the cochlea. The mass is strikingly irregular, bearing numerous projections. At the tympanic extremity, the sclerotic bone is exposd to the mucous membrane without the intermediation of cartilage or connective tissue; here, as well as at the vestibular extremity, the fissular channel is still lined by cartilage. Had the fissula in this specimen possessed an auxiliary, or fenestral, orifice (fig. 9) and had the new sclerotic bone followed such a preexistent channel, invading the vestibular window, fixation of the stapes would have resulted from impingement of the sclerotic bone on the stapedial base.

It is quite conceivable also that the newly formed cartilage (fig. 9), were it to protrude into the vestibular window, as it has in other specimens been observed to project into the semicanal of the tensor tympani muscle, might embarrass the stapes in its excursion; calcification of the tissue within the stapedial (annular) ligament would, of course, result in fixation of the stapes.

5. *Fossula post Fenestram.*—The fossula post fenestram is generally similar to the fissula ante fenestram in development and in structure (Bast, 1938[20]), but it is usually smaller. The fossula, according to Bast, is less frequent in occurrence than the fissula, being present in 67 per cent of the ears studied; in only 25 per cent of the ears possessing a fossula is the channel complete, i. e., extending through the wall of the otic capsule without interruption from the vestibule to the tympanic cavity. So far as observed the fossula does not display a similar variety of tissues: it does not contain activated cartilage or sclerotic bone. For

19. Wilson, J. G., and Anson, B. J.: Form and Structure of an Area of Otitic Sclerosis in the Temporal Bone of an Adult, Arch. Otolaryng. **18**:291-297 (Sept.) 1933.

20. Bast, T. H.: Development of the Otic Capsule: IV. The Fossula Post Fenestram, Arch. Otolaryng. **27**:402-412 (April) 1938.

this difference between the fissula ante fenestram and the fossula post fenestram we are unable to offer an explanation.

6. *Extracapsular Tissues.*—From measurements made on a collection of skulls we find a marked increase of the petrous portion of the temporal bone between fetal and adult stages; its length in the adult is one and a half times as great as that of the bone in the fetus at term; its width is virtually doubled. Thus, while the essential portions of the contained labyrinth remain unaltered after the fetal size is attained, the encapsulating bone undergoes marked increase in dimensions.

The enlargement of the petrous portion is due almost entirely to increment in tissues external to the otic capsule, since the "adult" dimensions of the combined cochlear and canalicular portions are attained in midfetal life. But between the later fetal and the early juvenile stages an extensive mass of pneumatic and marrow tissue is added, chiefly to each extremity of the primitive capsule, the addition at the anteromedial extremity (so-called apex or tip) being especially striking.[21] In a case already reported on, the air cells almost completely surround the carotid canal, extending from the very tip of the petrous portion to the cochlear area; in the latter situation they lie on both medial and lateral aspects of the cochlea, immediately against the bone of the otic capsule; communicating with the tympanic cavity on one side, they extend across the bone to its inner table on the opposite side. The highly trabeculate character of the bone and the vascular nature of the submucosal and marrow tissue no doubt account for the rapid extension of these extracapsular areas.[22]

CONCLUSIONS

The temporal bone, as a whole, exhibits a progressive set of changes, which continue through the life of the subject. They belong in two categories, growth and internal alteration in the otic capsule and the associated stapes.

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21. The air cells are abundant in the young child, indicating that development in early postnatal stages is rapid. At present we are studying these cells in representative specimens of several age groups; we have already presented an account of their wide distribution in the child of 4½ years (Anson, B. J.; Wilson, J. G., and Gaardsmoe, J. P.: Air Cells of Petrous Portion of Temporal Bone in a Child Four and a Half Years Old, Arch. Otolaryng. **27**:588-605 [May] 1938). Wax plate reconstructions are being prepared from represenative series in order to demonstrate the form and topographic relations of the cells satisfactorily.

22. The anatomy of the petrous portion of the temporal zone exclusive of the otic capsule and its contents has been presented in an excellent article by S. R. Guild (Normal and Pathological Anatomy of the Petrous Pyramid, Tr. Am. Otol. Soc. **25**:165-179, 1935); therein are considered the framework of the pars petrosa, the marrow and the air cells which are contained within it and the nerves and vessels which traverse it en route to destinations outside the temporal bone.

The several centers of ossification which give rise to the otic capsule have coalesced in the midfetal stage, yet the petrous portion continues to enlarge, pneumatic and marrow tissues being added after birth, the mass of the encapsulating element increasing while that of the contained labyrinthine core retains essentially fetal dimensions.

Similarly, the fenestral opening (vestibular window) in which the base of the stapes rests has attained adult dimensions during the first half of fetal life; so also has the stapes itself. Yet the rim of hyaline cartilage which lines the window is gradually reduced in breadth and thickness as age advances, being much thinner in the old adult than in the fetus of 4 or 5 months. Concurrently, the stapes is changed in early fetal stages from a cartilage having a solid base, crura and head to one of much more delicate structure, predominantly osseous—possessing crura of troughed form, an excavated head and a thin bilaminar base. Replacement of cartilage by bone then continues slowly throughout life.

These structural alterations in the window and the ossicle are accomplished without change in over-all dimensions. In the same category belong the fluctuations which mark the special area of the otic capsule situated between the vestibular window and the cochlea; here is situated the fissula ante fenestram, and here occur abnormal changes with such regularity that the area is referred to clinically as the "site of predilection" for otosclerosis. Although the entire region of the fiussla may not exceed 1 cu. mm., it is nevertheless the scene of histologic reconstruction profoundly important to the subject.

The fissula ante fenestram is typically an irregular channel through the temporal bone situated in front of the vestibular window; in the cleaned skull it is an open channel, admitting a bristle, but in the natural state it is occupied by fibrous tissue. Its immediate wall is formed by cartilage, which, like the cartilaginous content of the stapes or the chondral lining of the vestibular window, is a remnant of the originally larger collection of the same tissue, gradually encroached on by bone.

But the changes are not merely those of slow replacement of one tissue by another; certain other, more rapid alterations occur, which mark the area as histologically unstable. These seem to be related fundamentally to the secondary formation of hyaline cartilage, derived from the cartilaginous lining of the fissular channel. Displacing the fibrous tissue of the fissula, the nodule of newly formed tissue extends through the length of the original channel; it may not only extend to the regular tympanic and vestibular orifices but also occupy the auxiliary opening at the vestibular window. Under these circumstances the cartilaginous lining of the fissula and the encapsulating bone are the site of only the slightest activity. Here an intermediate tissue is present, derived from hyaline cartilage but usurped by bone; this tissue, formed initially as

cartilage, is invaded by osteoblasts, which convert the matrix into a calcified structure. Seemingly resistant to rapid change, this hybrid tissue ("intrachondrial bone" or "cartilage islands"), gradually incorporated into the wall of the fissula, remains readily distinguishable from the mass of activated cartilage, when such occurs; one is quiescent in character and constitutes part of the fissular wall; the other is active and is situated within the fissular channel; they seem independent, the less active being resistant to the more active tissue.

But within the fissular channel another type of tissue occurs which is sufficiently invasive to replace, in succession, any fibrous tissue remaining in the fissular channel, the newly formed cartilage located there, the older hyaline cartilage and intrachondrial bone of the fissular wall and finally the endochondral bone which immediately borders the fissular wall. This mildly invasive tissue, much more vascular than the other tissues of the otic capsule, spreads beyond the normal confines of the fissula, occasionally to reach the anterior wall of the vestibular window and to reduce the latter's space. After having taken the place of the hyaline cartilage which forms the primordial fenestral wall, the sclerotic bone, should it impinge on the base of the stapes, would fasten the ossicle within the window.

The total area affected by these processes is relatively small, so that an otosclerotic nodule of minute proportions may profoundly alter the equally small window and stapes; it is, so to speak, a pathologic process on a diminutive scale.

CONGENITAL CYSTS OF THE LARYNX

REPORT OF A CASE

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Congenital or embryonic cysts of the larynx are of interest to the laryngologist primarily because of their extreme rarity. In fact, any type of cyst is seen rather infrequently in this region. Of 722 cases of benign laryngeal tumors encountered at the Mayo Clinic prior to 1938, only 35 were cases of cysts, and in but 1 was the cyst of embryonic origin. This case was reported previously.[1] Since then we have encountered 2 other cases of congenital cysts of the larynx, of which we wish to mention 1 and to report the other at this time.

Cysts of the larynx are of three types, namely, mucous, hemorrhagic and congenital. Most common are the mucous cysts; their etiology undoubtedly points to an inflammatory basis which results in an obstruction of the ductal portion of a mucous gland. Because of this atresia, the mucus is unable to escape and accumulates within the glandular lumen. The effect of such a process is the production of a cyst. If the tumor does not become too greatly distended with mucoid secretion, the normal columnar or cuboidal glandular epithelium will be retained as the lining of the cyst. However, as the tension of the contained fluid increases, the epithelial cells become markedly flattened. With further expansion of the cyst, atrophic changes occur in the epithelium, which finally disappears altogether. This process affords an explanation as to why the great majority of mucous cysts, on removal, are found to be lined not with epithelium but with a smooth layer of fibrous connective tissue, the growth of which is stimulated by the irritative effects of abnormal tension. Although mucous cysts may occur anywhere within the larynx, they are encountered most frequently, as would be expected, in the vicinity of the epiglottis, where the mucous membrane is abun-

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1. (*a*) New, G. B.: Cysts of the Larynx, Journal-Lancet **37**:99-102 (Feb. 15) 1917. (*b*) New, G. B., and Erich, J. B.: Benign Tumors of the Larynx: A Study of Seven Hundred and Twenty-Two Cases, Arch. Otolaryng. **28**:841-910 (Dec.) 1938.

dantly supplied with mucous glands. Many mucous cysts in the larynx reach significant dimensions, but few, if any, attain the large proportions of a congenital cyst.

Although uncommon, hemorrhagic cysts occur much more frequently in the larynx than do those of embryonic origin. After an extravasation of blood into the tissues, if the blood is not absorbed, fibroblasts may proliferate and form a sort of connective tissue capsule around the clot; by this sequence of physiologic activities, a hemorrhagic cyst is developed. Such cysts are never lined with epithelium and usually contain coagulated blood or thin serous fluid with a small amount of fibrin. Hemorrhagic cysts, which are generally dark red or bluish, occur with few exceptions on the vocal cords. This is explainable by the fact that the vocal cords, more than any other part of the larynx, are subjected to traumatic injuries through overuse or misuse of the voice.

Laryngeal cysts of the mucous and hemorrhagic varieties are not true tumors; that is, they are composed of cells which fulfil no physiologic function. On the contrary, they represent the physiologic culmination of traumatic or inflammatory processes. Embryonic cysts in the larynx, on the other hand, are genuine neoplasms.

The etiology of these congenital cysts has attracted our attention. Many hypotheses have been advanced to explain their development. A number of years ago, Schneider [2] brought forth the hypothesis that such tumors arise from displaced embryonal cells which take part in forming the appendix of the ventricle, and Louys [3] expressed the opinion that they are derived from the mesodermal tissue of the branchial arches. After considerable study, we are inclined to believe that Schneider's hypothesis is correct. A microscopic examination of the wall of the congenital cyst which we removed revealed a lining composed of stratified columnar epithelium containing mucous glands, whereas the subepithelial tissues were markedly infiltrated with lymphocytes (fig. 1).

At first the question arose in our minds as to the probability of this cyst's actually being a branchial cyst, since the larynx is formed from the fourth and fifth branchial arches. It would appear logical that a branchial cyst could occur as readily mesial to the thyroid cartilage as externally. Microscopically, both types of cysts bear some points of resemblance, since both may be lined with stratified columnar epithelium. However, in the walls of branchial cysts there are aggregations of lymphoid tissue that contains germinal centers. In our laryngeal cyst, no such lymphoid tissue was present; only an infiltration of lymphocytes was found. On comparing the wall of the cyst with the wall of the appendix of the ventricle, we found that there was great similarity. Since the laryngeal appendix in man is a functionless and degenerative

2. Schneider, P., cited by New.[1a]
3. Louys, E., cited by New.[1a]

vestige of an important pouch in lower animals, it would not seem incredible that its development in the human being could be attended occasionally by the sequestration of some of its embryonal cells and that such cells, when segregated, could continue to multiply and eventually form a cyst. Then too, it is noteworthy that congenital cysts of the larynx always develop in close proximity to the laryngeal appendix, that is, in the lateral wall of the larynx or in the aryepiglottic fold.

Fig. 1.—The wall of a congenital laryngeal cyst (× 50). The lining is composed of stratified columnar epithelium and contains mucous glands. There is a lymphocytic infiltration in the subepithelial tissues.

Etiologically, one might consider a congenital laryngeal cyst analogous to inclusion dermoid cysts, which develop from inclusions of displaced dermal cells along the lines of embryonic fusion, such as the middorsal and midventral lines and the branchial clefts. In considering the situation from every angle, we find it most reasonable to assume that a congenital laryngeal cyst arises from cells sequestrated from the embryonic cells which take part in forming the appendix of the ventricle.

Although congenital laryngeal cysts have been observed in newborn infants, these tumors need not be present necessarily at birth. Some of them are first apparent in persons who have reached adult life. However, if small, these cysts may exist for many years without producing any noticeable symptoms. Generally their growth is extremely slow, but should one become infected, its dimensions may increase with alarming rapidity.

There are no symptoms diagnostic of a congenital cyst of the larynx. Patients having such a neoplasm may complain of none, one, some or all of the following symptoms: dyspnea, hoarseness, cough, desire to clear the throat and a sense of fulness in the hypopharynx. But these signs and physical complaints are not characteristic of a specific kind of tumor; they are, in fact, typical of almost any laryngeal lesion.

The diagnosis must be based on visual inspection of the larynx. These cysts are confined to the lateral wall of the larynx or bulge into the aryepiglottic fold. A few penetrate the thyrohyoid membrane and expand into the neck. Usually they have a smooth, shiny, semitranslucent, tense and cystic appearance. Blood vessels course over the surface of some. When touched with a probe, they are compressible. The nature of such cysts, if situated deeply in the lateral laryngeal wall, may not be obvious, since the structures composing the lateral wall are merely pushed into the lumen of the larynx. Many congenital cysts that involve the aryepiglottic fold are of such large dimensions as to block completely a view of the glottis. With some such cysts it is amazing to observe the small amount of respiratory difficulty. This, of course, is based on the very gradual increase in the size of the cyst, which obstructs the larynx so slowly that the patient is entirely unaware of and becomes accustomed to the diminishing volume of air. Were the same degree of obstruction to occur within a few hours or days, the patient would require a tracheotomy as an emergency measure.

In most instances, the diagnosis is comparatively simple, although some of the cysts can be confused with other laryngeal lesions. A few years ago a patient was seen at the clinic with a smooth, rounded tumor involving the right aryepiglottic fold. On indirect laryngoscopic examination, the tumor appeared to be a cyst, but on further study it proved to be a lymphosarcoma. Congenital and mucous cysts deep in the lateral wall of the larynx or in the aryepiglottic fold can be identified only by microscopic examination. However, mucous cysts are uncommonly encountered in these situations.

The treatment of embryonal laryngeal cysts is dependent on their size and condition. When small they may be removed under direct laryngoscopy, the Lynch suspension apparatus being employed, but when large they can be exposed and excised only by means of thyrotomy. Patients who are severely dyspneic may require a preliminary trache-

otomy before any type of treatment is undertaken. When such a cyst is infected and contains purulent material, it may be advisable to incise it under suspension laryngoscopy, thus allowing its fluid contents to be expelled and permitting its walls to collapse. Frequently the cavity of the cyst subsequently will become obliterated by the formation of scar tissue. However, if the cyst tends to refill, it can be excised safely after the acute inflammatory reaction has subsided.

The first congenital laryngeal cyst seen at the clinic has been reported previously in our series of 722 cases of benign tumor of the larynx.[1b] It occurred in a girl aged 14. Soon after her birth, stridor was noted. Hoarseness and dyspnea occurred later. When she was 7 the cyst was discovered, incised and drained several times, but there was no permanent relief of her symptoms. Examination at the clinic revealed a hemorrhagic mass in the right aryepiglottic fold. Under suspension laryngoscopy, the cyst was incised and drained, with evacuation of 1½ ounces (44 cc.) of pus. Drainage was followed by healing, fortunately, and the patient has not had further trouble. Tissue was not obtained for microscopic study because the lining of the cyst had been destroyed by the infection, which had been present for years.

The second congenital cyst which we encountered did not cause symptoms and was not visible clinically. It occurred in a man of middle age who had an extensive epithelioma of the vocal cords. Situated deep in the left lateral wall of the larynx, the cyst penetrated the thyrohyoid membrane and bulged into the neck, much as in the first of the three cases described and illustrated by Davis.[4] This cyst was not discovered until its finger-like projection, external to the thyrohyoid membrane, was cut across while a laryngectomy was being performed for the removal of the malignant growth. Because of the small dimensions of the intralaryngeal portion of the cyst, it did not disturb the normal contour of the interior of the larynx sufficiently to make it noticeable on laryngoscopic examination.

The third congenital laryngeal cyst occurred in the following case:

A 17 year old girl had been hoarse since she was first able to talk. Because this symptom became progressively more severe, she finally consulted an otolaryngologist, who discovered a tumor in the hypopharynx. He advised her to come to the clinic as soon as her school term was ended. Accordingly, three months later, she registered in our section for consideration.

On indirect laryngoscopic examination, a large, smooth, rounded, tense and cystlike mass was found bulging the right aryepiglottic fold, involving the right wall of the larynx and extending into the right piriform fossa (fig. 2). A view of the glottis was impossible because of the large size of the growth. With such a limited airway, it seemed remarkable that the patient did not complain of dyspnea.

4. Davis, E. D. D.: Cysts of the Larynx, J. Laryng. & Otol. **38**:473-476 (Sept.) 1923.

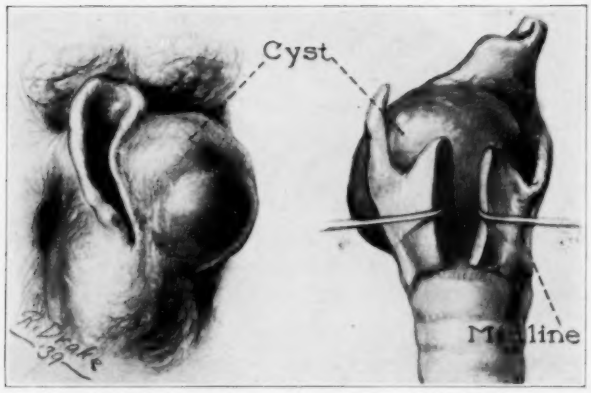

Fig. 2.—At the left, the congenital laryngeal cyst, as seen from its posterior surface. The cyst was situated deep in the right lateral wall, markedly bulged the right aryepiglottic fold so as to obstruct the supraglottic region of the larynx almost completely and extended into the right piriform fossa. At the right, the relative position of the congenital laryngeal cyst in relation to the epiglottis, the right aryepiglottic fold and the thyroid cartilage. The right lamina of the thyroid cartilage has been divided by a method similar to that carried out during the operation described.

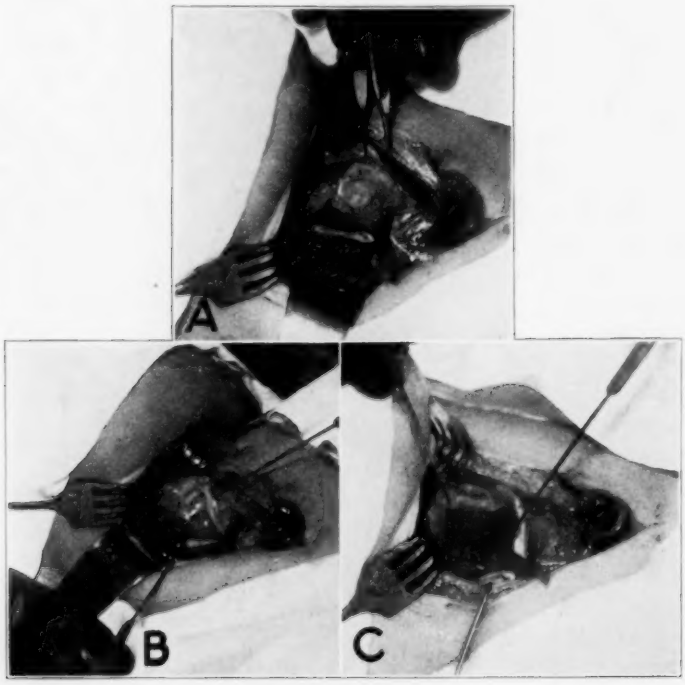

Fig. 3.—Photographs taken during the operation for the removal of the congenital laryngeal cyst. The patient's chin is at the left, and the tracheal tube is at the right. *A*, the tip of the forceps is in the thyroid notch, which lies in the median line. The right lamina of the thyroid cartilage has been divided for exposure of the cyst. *B*, the hooks retract the cut edges of the cartilage and expose the underlying cyst. *C*, the two hooks on the right retract the cut edges of the thyroid cartilage; the hook on the left retracts the thyrohyoid membrane. The cyst has been completely removed, and the large remaining pocket is visible.

A preliminary diagnosis of congenital laryngeal cyst was made with the recommendation that a tracheotomy be performed, to be followed by an exploration of the mass.

Routine examinations of the blood and the urine gave essentially negative results, and her general physical condition was good. Roentgenologic examination of the cervical region showed some narrowing of the retrolaryngeal soft tissue space.

Four days after the patient's arrival, the trachea was opened just below the cricoid cartilage and a tracheal cannula was inserted. This procedure was absolutely necessary because of the large size of the growth, which undoubtedly would have obstructed the larynx completely during the subsequent manipulations employed in examining and removing the cyst.

Twelve days after the tracheotomy, the patient was anesthetized with nitrous oxide gas and ether, and a direct laryngoscopic examination was performed, the Lynch suspension apparatus being used. Even with this method for obtaining a direct view of the larynx, the cyst was entirely too large to allow visualization of the glottis. It was then decided that removal of the cyst under suspension laryngoscopy would be inadvisable and that a thyrotomy was indicated.

Consequently, after two days had passed a thyrotomy was undertaken. A cutaneous incision was made in the midline, and the right lamina of the thyroid cartilage was divided in a vertical direction, 1 to 2 cm. to the right of the junction of the two thyroid laminas (fig. 2 and 3*A*). The cyst was exposed and was found to bulge above the thyroid cartilage (fig. 3*B*). By careful dissection, the surgeon removed the entire tumor without cutting through or into the mucosa of the larynx. At one point the cyst was accidentally nicked, and a thin whitish fluid was discharged. However, this rupture did not interfere with complete excision. No definite connection of the cyst with the ventricle or its appendix was discovered. The two portions of the thyroid cartilage were reunited with interrupted sutures of chromic gut, and the overlying skin was approximated with sutures of fine black silk. Two Penrose drains were inserted; one was arranged above the thyroid cartilage so as to extend into the pocket formed by the removal of the cyst (fig. 3*C*), and the other was placed lateral to the right lamina of the thyroid cartilage.

The patient made an uneventful recovery and was discharged from the hospital nineteen days later, with the tracheal tube removed. After another three days, she was dismissed from the clinic. The hoarseness entirely disappeared, and the internal aspect of her larynx became perfectly normal.

TREATMENT OF OTITIC MENINGITIS

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For many years meningitis has been accepted as one of the complications of purulent disease of the middle ear, and it is one of the most dreaded complications which may follow any surgical procedure. Since a really scientific interpretation of disease of the middle ear and mastoid has been recognized, attempts at treatment and cure of meningitis have been pathetically disappointing. The entire gamut of surgical, medical and chemical procedures has been tried, and temporary enthusiasm has given way to extreme pessimism as statistics showed that no progress was being made. The one exception is that radical surgical intervention plus spinal drainage seemed to give a few cures, extremely few on the basis of percentage. Gray surveyed the literature from 1901 to 1935 and collected 2,200 cases of otitic meningitis with only 66 recoveries, a mortality of 97 per cent. Neal, in the New York Department of Health, collected 238 cases from 1910 to 1935, with a mortality of 95 per cent. (This, by the way, was the best showing of cures in any series reported.) From 1920 to 1935, at Johns Hopkins Hospital, not a single patient with otitic meningitis recovered. At the Manhattan Eye, Ear and Throat Hospital, from 1926 to 1936, 101 cases of meningitis were reported, with 2 recoveries, a mortality of 98 per cent.

It is my intention to describe the treatment and results in 14 consecutive cases of otitic meningitis and 1 case of meningitis following submucous resection in which admission was made to my service at Queens General Hospital during the past two years. Only the cases in which the organism was actually recovered in the spinal fluid are reported. In this series, treatment was comparatively similar in each case, namely, a combination of radical surgical intervention, daily spinal drainage and the intensive use of sulfanilamide or one of the related compounds. Clinical adjuncts, such as blood transfusions and intravenous administration of dextrose were employed during the same period.

TREATMENT

1. *Surgical Procedure.*—After a thorough simple mastoidectomy was performed, the dural plate over the temporal lobe was removed; the diameter of the bone removed in the average case was approximately

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1½ inches (4 cm.). The wound was filled with a 70 per cent solution of alcohol, which was allowed to remain in it for about two minutes. The cavity was then dried and the dura slit with three or four parallel incisions, free flow of the spinal fluid thus being allowed. The external ear was sutured forward and a doughnut dressing applied behind it, so that when pressure was applied during the bandaging the cutaneous surface could not adhere to the incisions in the dura and free drainage was thus not interfered with.

2. *Palliative Measures.*—On the patient's return to his room the foot of his bed was elevated 18 inches (46 cm.), and continuous intravenous administration of a 10 per cent solution of dextrose by means of the old-fashioned Murphy drip was maintained for several days to prevent dehydration. As many transfusions were given as deemed necessary for the patient's general condition, probably an average of six in each case, the amount of blood depending on the age of the patient. Daily spinal taps were done to flush out the meninges, to reduce the pressure of the spinal fluid and to examine it for organisms and cell count.

3. *Sulfanilamide Therapy.*—Sulfanilamide and neoprontosil were given in intensive doses, by mouth, intramuscularly and intraspinally, the dose of each depending on the age and weight of the patient. In the use of sulfanilamide and neoprontosil it is important to produce a high concentration of the chemotherapeutic drug in the blood as soon as possible and to maintain this concentration as long as necessary. Therefore, during the first forty-eight hours, it is my custom to administer the drug to the limits of tolerance, and at the end of that time, as a rule, the amount is cut down, as the acute symptoms have somewhat subsided. Absorption of the drug is rapid, as sulfanilamide, the form given by mouth, is almost completely absorbed in four hours, and therefore the drug should be given orally within four hours. It takes about forty-eight hours to obtain its maximum therapeutic effect. After the first forty-eight hours, the dose was cut down, but no matter how well the patient seemed small doses were given for at least one month.

In several of the cases to be reported, the spinal fluid was sterile for days, and unexpectedly the organism reappeared. Therefore, one should not stop giving the drug altogether for at least one month.

Dosage: As the condition in all the cases was severe, the doses were large at all times. During the first forty-eight hours, adults weighing 120 pounds (54 Kg.) or over received 1 cc. of neoprontosil for each pound (0.5 Kg.) of body weight per day, i. e., 20 cc. of neoprontosil each four hours, and, in addition, 20 grains (1.3 Gm.) of sulfanilamide by mouth each four hours and 0.8 Gm. of sulfanilamide powder in 100 cc. of saline solution injected into the spinal canal once or twice a

day according to the severity of the illness. The more spinal fluid withdrawn, according to the pressure, the more sulfanilamide solution was injected.

After the first forty-eight hours, if the patient showed improvement, the dose was cut down; if not, the same dose was kept up for twenty-four to forty-eight hours more. For children the dose was cut down according to their weight, regarding 120 pounds (54 Kg.) as the adult weight.

If oral administration alone was used, much larger doses were given, even as high as 50 to 80 grains (3.2 to 5.2 Gm.) for the initial dose, followed by 20 grains (1.3 Gm.) each four hours, for a person weighing 100 pounds (45 Kg.) or more.

The drug seems to act best when administered orally; but when the patient is unconscious and cannot swallow the administration of neoprontosil, as well as the intraspinal medication, may be greatly increased.

Personally, I feel that the drug should be administered in all three ways.

I met with no untoward results except slight cyanosis, dizziness, nausea and tinnitus at times, and on reduction of the dose all cleared up.

Daily estimations of hemoglobin and leukocyte and differential cell counts were made. One of the dangers of too large doses of sulfanilamide is the production of hemolytic anemia. The hemoglobin content has been known to drop from 100 to 18 per cent in a few days, and some cases have been reported in which agranulocytosis followed the use of the drug. These conditions may come on at any time up to a week after its administration is discontinued, which makes it important to continue daily blood counts for a week or ten days after that time. The concentration of the drug in the blood can be readily determined at the present time, and it is important to do this frequently, as a greater concentration is required to control some infections than others. The average infection is controlled by a concentration of 8 to 10 mg. per hundred cubic centimeters of blood. For a severe infection, such as meningitis, the concentration should be 15 mg. The amount which must be administered to attain a given concentration depends on the ease and rapidity with which a particular patient eliminates the drug. The fluid intake should be restricted to hasten the concentration.

BACTERIOLOGIC CLASSIFICATION

The bacteriologic agents in the present series of cases may be divided into four groups: (1) streptococcus, (2) pneumococcus, (3) staphylococcus and (4) Bacillus influenza.

Therapeutically and from the standpoint of prognosis, this grouping is important, as by far the best results were attained with the strepto-

coccic infections, from which 7 of 8 patients recovered. With the pneumococcic infections, on the other hand, 3 of 5 patients died. Unfortunately, these cases came under observation before sulfapyridine (2-[paraminobenzene sulfanimido]-pyridine) came into existence.

One patient infected with Staphylococcus albus, 1 with Pneumococcus type IV and 1 with Pneumococcus type XXIX recovered. One infection in the series, which came under observation three months ago, was caused by the B. influenzae. The patient was treated with a combination of sulfanilamide and sulfapyridine and recovered.

I should like to report a typical case of infection with Streptococcus haemolyticus, as it illustrates the necessity of surgical intervention in some cases. There were 4 such cases in the series, in which I believe the patients would not have recovered with the use of sulfanilamide alone, as clinically and by bacteriologic tests they failed to improve until surgical treatment was instituted, after which the change was so marked that it seems impossible to consider it a coincidence.

CASE 1.—A boy aged 8 years was admitted to the hospital on Jan. 28, 1937, with acute mastoiditis on the right. Simple mastoidectomy was done on February 1. He was discharged from the hospital on February 9. He did well at home for two weeks; then palsy of the right external rectus muscle developed, accompanied by a temperature of 103 F. He was readmitted to the hospital the following day, complaining of a general headache and pain in the ear.

At the physical examination the child appeared acutely and critically ill, with a temperature of 105 F., a stiff neck, double Kernig signs and pallor of both optic disks. The spinal fluid contained 820 cells, with 65 per cent polymorphonuclears, and yielded a positive culture for Str. haemolyticus. Administration of sulfanilamide in large doses, by mouth and intramuscularly, was started at once, on February 24.

The patient remained acutely ill for the following three days, with a temperature of 105 to 106 F., severe headache and palsy of the external rectus muscle. On February 27 the petrous apex was explored, but a fistulous tract or other evidence of pathologic change could not be found. Postoperatively the temperature remained septic, ranging between 101 and 105 F. daily. Repeated blood cultures were negative. This continued until March 10, when the lateral sinus was exposed for possible sinus thrombosis. It appeared normal and was not opened. The child continued to do badly, having an irregular temperature curve. The palsy of the rectus muscle and the ptosis remained almost the same; meningeal signs were increased; nuchal rigidity was more marked, and a 4 plus Kernig sign was present. The spinal fluid continued to be cloudy, with a high cell count and hemolytic streptococcus still present. This condition prevailed in spite of intensive sulfanilamide and neoprontosil therapy. Numerous intravenous infusions of a 10 per cent solution of dextrose and four blood transfusions of 300 cc. each were given. Spinal taps were done daily to relieve the pressure. Culture of the spinal fluid was positive every day. The child continued to decline, and the situation appeared desperate.

On March 29, thirty-three days after the second admission to the hospital, he was again taken to the operating room, and the dura over the temporal lobe was

slit horizontally by three parallel incisions, each about ¾ inch (2 cm.) long, to allow for free drainage of the spinal fluid. The child was then placed in bed, the foot being elevated 18 inches (46 cm.).

From this point on the child began to improve. The temperature curve began to flatten out, although it reached 101 or 102 F. daily. The child seemed much improved clinically, with marked improvement in the palsy of the rectus muscle and the ptosis. He seemed to be less drowsy and took nourishment better. The spinal fluid rapidly cleared after the operation, and four days later the culture was negative for the first time, after twenty-four consecutive positive cultures, with a cell count of only 150 and the sugar content increasing.

The dural wound drained freely for several days. Herniation of the brain did not occur. Two months after operation the child's temperature was normal, and the wound had filled with granulation tissue. Culture of the spinal fluid was negative, and the only residue of the illness was slight spasticity of the lower right extremity. Administration of sulfanilamide in decreased doses was continued. On June 15 the child was discharged well.

The data on the spinal fluid throughout the illness were as follows:

Date, 1937	Culture	Cell Count
February 26	Positive, Str. haemolyticus	820
February 28	Positive, Str. haemolyticus	1,450
March 1	Positive, Str. haemolyticus	2,680
March 2	Positive, Str. haemolyticus	1,900
March 3	Positive, Str. haemolyticus	1,640
March 4	Positive, Str. haemolyticus	920
March 5	Positive, Str. haemolyticus	1,118
March 6	Positive, Str. haemolyticus	1,040
March 7	Positive, Str. haemolyticus	2,155
March 9	Positive, Str. haemolyticus	2,300
March 11	Positive, Str. haemolyticus	1,060
March 15	Positive, Str. haemolyticus	725
March 17	Positive, Str. haemolyticus	1,010
March 18	Positive, Str. haemolyticus	1,450
March 19	Positive, Str. haemolyticus	950
March 20	Positive, Str. haemolyticus	1,006
March 22	Positive, Str. haemolyticus	725
March 23	Positive, Str. haemolyticus	1,010
March 25	Positive, Str. haemolyticus	1,450
March 26	Positive, Str. haemolyticus	950
March 28	Positive, Str. haemolyticus	760
March 30	Positive, Str. haemolyticus	450
April 2	Sterile	220
April 5	Sterile	150
April 10	Sterile	108
April 15	Sterile	28

A résumé of this case strongly suggests that the various forms of sulfanilamide are insufficient to effect a cure, as was shown by the continuance of meningitic symptoms and the constant finding of hemolytic streptococcus in the spinal fluid. The real change for the better seems to have started immediately after decompression with dural slits to allow free drainage of the spinal fluid.

There were 4 deaths in the series, 3 caused by pneumococci and 1 by Str. haemolyticus. The last followed the complication of an abscess of the temporal lobe, which unfortunately was not recognized in time. One infection with Pneumococcus type III, in which the course was rapid and fulminating, appeared hopeless from the beginning and termi-

nated fatally within forty-eight hours. The other 2 fatal infections were with Pneumococcus type I and had several points in common:

1. The onset and course were extremely rapid.

2. Local symptoms of danger were outwardly lacking, as the drum membrane appeared almost normal, sagging of the wall of the canal was not present and the usual symptoms of mastoiditis requiring surgical intervention were missing. Regardless of these findings, mastoidectomy revealed almost complete necrosis of the entire mastoid cavities, including the sinal and dural plates.

3. Death resulted in both cases even though intense surgical and medical endeavors were instituted.

At this time it seems logical to stress the importance of the newer drug, sulfapyridine, for it is well known that it is particularly efficacious against the pneumococcus group, and if it had been known at the time when these infections occurred it is easily possible that they too might have terminated in recovery.

REPORT OF ADDITIONAL CASES

CASE 2.—E. B., a 4 year old girl, entered the hospital on March 16, 1937. Her past history was irrelevant. Two weeks prior to admission a high temperature suddenly developed, accompanied by headache and pain in both ears, which persisted up to the time of admission to the hospital. On March 10, 1937, six days before admission, the left ear spontaneously ruptured. Physical examination revealed an acutely ill child, with a temperature of 105 F., a pulse rate of 132, restlessness, severe headache, vomiting and stiffness of the neck.

The left ear drum was red, full and bulging, with the landmarks obliterated. Some dry exudate was present. Slight auricular edema was present, as well as suggestive tenderness over the mastoid. Myringotomy was performed and thick pus obtained. The right ear drum was injected but otherwise normal.

Neurologic examination showed definite nuchal rigidity, with positive Kernig and Babinski signs. Palsy of the sixth or seventh nerve and nystagmus were not present. A spinal tap revealed slightly opalescent fluid under a pressure of 250 mm. of water; the cell count was 1,200, with 64 per cent polymorphonuclears and 36 per cent lymphocytes. No organisms were seen on smear, but culture showed Str. haemolyticus. The leukocyte count was 26,000, with 86 per cent polymorphonuclears, 10 per cent lymphocytes and 4 per cent transitional cells. Mastoidectomy was done on the left the same day. The cells were hemorrhagic, with occasional beads of pus. However, there was extensive softening and destruction of all the cells. The sinal plate was removed, and the sinal wall appeared healthy. The dura of the middle fossa was exposed during the operation. Culture of material from the mastoidectomy wound showed hemolytic streptococci. Large doses of sulfanilamide and neoprontosil were prescribed.

The following day, when the report on the culture of the spinal fluid was available, temporal decompression was performed, and the dura over the temporal lobe was slit in several places (three or four parallel incisions). For three days after operation the temperature ranged between 105 and 106 F., and then it gradually came down.

A résumé of the case follows.

March 16, 1937: admission to the hospital; simple mastoidectomy on the left; administration of sulfanilamide, two 5 grain (0.32 Gm.) tablets each four hours for forty-eight hours; neoprontosil, 1 ampule each six hours, intramuscularly and sulfanilamide crystals, 0.8 Gm. in saline solution, intraspinally; blood transfusion, 300 cc.; continuous intravenous administration of 10 per cent solution of dextrose.

March 17: operation uncovering temporal dura and making several slits in it, to allow free flow of spinal fluid; elevation of foot of bed 18 inches (46 cm.); intravenous administration of dextrose continued; blood transfusion, 300 cc.

March 18: reduction of dose of sulfanilamide to one 5 grain (0.32 Gm.) tablet each four hours; administration of neoprontosil, 1 ampule each six hours, and of sulfanilamide crystals intraspinally continued.

March 19: blood transfusion, 300 cc.

March 21: absence of meningeal signs, headache and rigidity; temperature, 101 F.

March 29: administration of sulfanilamide and neoprontosil in same doses continued.

April 11: reduction of dose of sulfanilamide to one 5 grain (0.32 Gm.) tablet each eight hours.

May 5: patient discharged well.

The data on the spinal fluid throughout the illness were as follows:

Date, 1937	Culture	Cell Count
March 16	Positive, Str. haemolyticus	1,200
March 17	Positive, Str. haemolyticus	1,618
March 18	Positive, Str. haemolyticus	2,100
March 19	Sterile	1,400
March 24	Sterile	1,260
March 27	Sterile	260
April 1	Sterile	10
April 4	Sterile	4
April 11	Sterile	4

Apparently the invasion of the meninges occurred at the outset of the illness, when the patient became suddenly ill, had a temperature of 105 F. and complained of headache and pain in the ears. On the day of admission to the hospital, March 16, 1937, the patient had full-blown meningitis.

Case 3.—M. S., a 24 year old white typist, unmarried, whose past history was irrelevant except for a miscarriage three years previously, was admitted to the hospital on March 18, 1937. Three weeks prior to her admission a cold associated with frontal headache and pain in the right ear had developed. The ear spontaneously ruptured and discharged up to the time of admission. The headache had become increasingly severe, radiating to the occipital region, in the past two weeks. The temperature ranged to 102 F. For the past two months the patient had been amenorrheic.

Physical examination revealed an acutely but not critically ill woman, with a temperature of 100 F., a pulse rate of 60 and a respiratory rate of 20.

In the right ear the hearing was impaired to perception of a whisper at 6 inches (15 cm.) with a Bárány noise apparatus in the left ear. The drum was reddened and edematous, with a scant thin discharge which did not reappear rapidly when wiped away. Sagging of the canal wall and tenderness over the mastoid were not present. The left ear revealed only redness of Shrapnell's membrane. Neuro-

logic examination on admission gave negative results. Meningeal signs, nystagmus and involvement of the sixth or seventh nerve were not present. The fundi were normal. The nose was normal, without polypi or pus. The heart and chest were normal. That afternoon the temperature rose to 103 F., and the patient appeared to be in a more toxic condition. In view of the history of a previous miscarriage, the vagina was examined. The cervix seemed soft, suggestive of Hegar's sign, and the fundus seemed enlarged. The possibility of a self-induced abortion with sepsis was entertained.

The next day, however, the temperature went to 105 F., with increased headache and stiffness of the neck. A spinal tap revealed cloudy fluid under increased pressure (500 mm. of water); the cell count was 4,000, all polymorphonuclears. Culture showed type III pneumococci. The blood count was 20,000, with 97 per cent polymorphonuclears.

Simple mastoidectomy was performed on the right. A large pneumatic mastoid was encountered, all cells being necrotic and filled with granulation tissue, but no frank pus was found. Pathologic exposure was not noted. The wound was left open. Culture of material from the mastoid was reported to be sterile. Intensive sulfanilamide and neoprontosil therapy was initiated, but the next day the patient died. Autopsy showed mastoiditis on the right, purulent meningitis, pericardial effusion and local venous thrombosis, with myocarditis and congestive sphenitis.

The data on the spinal fluid were as follows:

Date, 1937	Culture	Cell Count
March 20.....	Positive, Pneumococcus type III	4,000
March 21.....	Positive, Pneumococcus type III	6,400

The bacterial findings in this case are interesting in that Pneumococcus type III, otherwise known as Streptococcus mucosus encapsulatus, while an extremely virulent organism, is nevertheless nonhemolyzing. Sulfanilamide is supposed to be most efficacious, or efficacious only, against hemolyzing organisms, and it is possible that had the organism been a hemolytic streptococcus or had sulfapyradine been available at the time the outcome might have been different. The history of pregnancy and amenorrhea unfortunately caused extreme confusion in the diagnosis and delay in surgical intervention.

CASE 4.—W. H., a 7 year old white boy, on the day prior to his admission to the hospital complained of headache, vomiting and pain in both ears. The next day a punctate erythematous rash appeared over his body, and he was admitted to Queensboro Pavillion, the unit for contagious diseases of Queens General Hospital, with a conjectural diagnosis of scarlet fever, on March 24, 1937.

When first seen, he appeared moderately ill. Over the trunk and body the rash appeared. Both ears were red and full, with the landmarks obliterated. The tonsils were enlarged, with some cervical adenopathy; otherwise the physical examination gave negative results. A diagnosis of scarlet fever and bilateral acute purulent otitis media was made.

The child continued to have fever for ten days after admission. He had extreme tenderness over the left mastoid. Simple mastoidectomy was performed on the left. Free pus was present in the initial groove. Culture showed this to contain Str. haemolyticus. There was moderate destruction of bone, especially in the zygomatic area. Pathologic or operative exposure was not recorded.

Postoperatively the child's temperature was elevated, ranging from 101 to 103 F. Tenderness over the right mastoid and aural discharges appeared. Seven days after operation palsy of the left external rectus muscle was evident. Roentgenograms of the petrous tips were reported normal.

On April 28 simple mastoidectomy was performed on the right. Free pus was found in the initial groove, and much cellular breakdown was present. The sigmoid sinus was exposed at operation. Str. haemolyticus was recovered on culture.

On May 1 the child had a chill; the temperature rose to 105.5 F., and he appeared acutely ill. A spinal tap was done, and a culture of Str. haemolyticus was reported, although neurologic signs were not present. Sulfanilamide and neoprontosil therapy was immediately instituted, 15 grains (1 Gm.) of sulfanilamide each four hours for the first forty-eight hours, followed by 10 grains (0.6 Gm.) each four hours, and 1 ampule of neoprontosil each six hours. Eight-tenths gram of sulfanilamide crystals in 100 cc. of physiologic solution of sodium chloride was injected intraspinally at this time. A transfusion of 300 cc. of blood was given.

On May 5 the child complained of pain behind the eye. The petrous tips were submitted to an additional roentgen examination, which was reported as follows: There is a suggestion of diffuse rarefying osteitis in the apical portion of the left pyramid, but the findings are not sufficient to lend any degree of certainty to the diagnosis of petrous destruction.

On May 11 positive signs of meningitis were present, namely, stiffness of the neck, bilateral Kernig signs and transient ankle clonus on the left. The spinal fluid, on culture, revealed Str. haemolyticus. The dura of the temporal lobe was uncovered and slit in four places.

On May 15 the patient was much better, and the spinal fluid was sterile.

On May 22 the palsy of the rectus muscle was improved.

On May 29 the nuchal rigidity and other meningeal signs had disappeared. Administration of neoprontosil was discontinued, but the patient was still receiving sulfanilamide by mouth.

On June 2 a transfusion of 300 cc. of blood was given.

On June 5 the temperature was normal.

On July 4 the patient was discharged well.

The data on the spinal fluid throughout the illness were as follows:

Date, 1937	Culture	Cell Count
May 1	Positive, Str. haemolyticus	400
May 2	Sterile	240
May 3	Sterile	280
May 11	Positive, Str. haemolyticus	120
May 12	Positive, Str. haemolyticus	910
May 13	Positive, Str. haemolyticus	740
May 15	Sterile	12
May 20	Sterile	1,000
May 21	Sterile	2,400
May 26	Sterile	750
May 28	Sterile	102
June 2	Sterile	30

This case appears to be a repetition of case 1; sulfanilamide alone was not sufficient to effect a cure. Although the symptoms were temporarily abated, they recurred more intensely than ever ten days after the institution of sulfanilamide therapy, but they were promptly and permanently removed after temporal decompression with dural slits.

CASE 5.—R. N., a boy aged 8 years, was admitted to the hospital on April 4, 1937, with the following history: March 30, 1937, fever and earache on the left; March 31, spontaneous rupture of the left ear drum; April 1, elevation of temperature and cessation of drainage from the ear; April 3, pain in the left ear, myringotomy done by the family physician, followed shortly by severe generalized headache, vomiting and dizziness; April 4, admission to the hospital.

Physical examination revealed an acutely and critically ill boy, with full-blown meningeal signs, headache, stiffness of the neck, a temperature of 106 F., bilateral Kernig signs and definite nystagmus to the right. Palsy of the left external rectus muscle was present. The left ear had a red, full drum, with no landmarks, and a moderate thin and mucoid discharge, without tenderness over the mastoid. The patient was irrational, and it was impossible to ascertain the presence of hearing. A spinal tap yielded 60 cc. of cloudy fluid, under pressure of 270 mm. of water, with a cell count of 2,030, with 100 per cent polymorphonuclears. A smear showed gram-positive diplococci in chains. Culture revealed the organism to be Pneumococcus type I. The blood count was 15,050, with 88 per cent polymorphonuclears, 8 per cent lymphocytes and 4 per cent eosinophils.

Roentgen examination of the mastoid was attempted but was unsuccessful, because the patient could not cooperate.

The same day simple mastoidectomy with temporal decompression and slitting of the dura in several places was performed on the left. Frank pus was found in the initial groove, with complete necrosis of all mastoid cells.

The following therapy was instituted: intensive intravenous and intraspinal administration of antipneumococcus serum, 1,250,000 units in forty-eight hours; administration of sulfanilamide tablets, 15 grains (1 Gm.) each four hours, and 1 ampule of neoprontosil each six hours; intraspinal injection of sulfanilamide crystals in saline solution; daily spinal taps, and repeated blood transfusions.

The child remained alive for thirty days, most of the time semicomatose and irrational, with a temperature ranging up to 105 F. daily. The spinal fluid became sterile on four different days but did not remain so. The palsy of the rectus muscle disappeared. At no time was there more than a gleam of hope, and finally the child died, on May 3.

Twenty-four spinal taps were done during the patient's stay in the hospital; on twenty-three of these occasions the fluid was examined, and the data were as follows:

Date, 1937	Culture	Cell Count
April 4	Positive, Pneumococcus type I	2,030
April 6	Positive, Pneumococcus type I	3,215
April 7	Positive, Pneumococcus type I	800
April 8	Sterile	880
April 9	Positive, Pneumococcus type I	1,460
April 12	Positive, Pneumococcus type I	3,820
April 13	Positive, Pneumococcus type I	1,170
April 14	Positive, Pneumococcus type I	660
April 16	Positive, Pneumococcus type I	1,332
April 18	Positive, Pneumococcus type I	1,200
April 19	Sterile	1,000
April 20	Positive, Pneumococcus type I	2,900
April 21	Sterile	360
April 22	Sterile	460
April 23	Positive, Pneumococcus type I	2,448
April 24	Positive, Pneumococcus type I	360
April 24	Positive, Pneumococcus type I	1,300
April 25	Positive, Pneumococcus type I	1,830
April 25	Positive, Pneumococcus type I	1,836
April 26	Positive, Pneumococcus type I	1,200
April 27	Positive, Pneumococcus type I	270
April 28	Positive, Pneumococcus type I	1,476
April 29	Positive, Pneumococcus type I	1,152

To digress for a moment, it seems rather conclusive that the meningitic invasion was the direct result of the myringotomy, since nystagmus, vomiting and dizziness appeared almost immediately after the drum was opened.

This was 1 of 2 cases in the series in which the organism was Pneumococcus type I, in both of which mortality resulted regardless of treatment. It therefore seems that this organism must be regarded as an extremely fulminating one, but the outcome might possibly have been different had sulfapyridine been in use at the time.

Case 6.—A. D., a 5 year old Italian girl, was admitted to the hospital on April 9, 1937, with a history of bilateral aural discharge of two and one-half weeks' duration. Examination revealed both drums red and bulging. A pulsating, thick discharge and suggestive sagging of the wall of the canal were present in the left ear. Tenderness over the mastoid was not present. The results of neurologic examination and those of physical examination other than the observations mentioned were negative. The urine contained albumin (2 plus) with many clumps of white cells. A diagnosis of bilateral acute purulent otitis media and pyelitis was made. During the next ten days the child had slight elevations of temperature, never over 101 F., and the urine cleared up. However, on April 19, ten days after admission, postauricular edema appeared on the right side. A profuse, thick, purulent discharge was present, which reappeared rapidly after being wiped away. There was definite sagging of the wall of the canal but still no tenderness over the mastoid. Roentgenograms revealed bilateral purulent mastoiditis with probable absorption of bone and perisinal abscess on the right side.

On April 19 simple mastoidectomy was performed on the right. At operation, extensive destruction of the mastoid was encountered, with frank pus in the initial groove. The dura of the middle fossa and the sigmoid sinus was pathologically exposed. The sinus was uncovered from the knee to the tip. Bone over the dura was removed until healthy dura was encountered. The wound was packed with iodoform gauze and left open. A culture of pus from the mastoid was reported to be sterile.

Postoperatively the child did not do well. The temperature continued to be elevated; she appeared pale and washed out, in spite of several transfusions. The urinary abnormalities reappeared. Ten days after operation stiffness of the neck and body was noted, and a spinal tap was done, on April 29, revealing cloudy fluid under a pressure of 230 mm. of water, containing sugar (1 plus) and globulin (2 plus), with a cell count of 4,280, with 91 per cent polymorphonuclears. No organisms were seen on smear; culture, however, revealed Pneumococcus type IV. Intensive sulfanilamide and neoprontosil therapy was instituted, but the child did not do well.

On April 30 the left mastoid was exposed and found to be normal.

On May 9 temporal decompression was undertaken, and dural slits were made.

The patient was subjected to daily spinal taps and several blood transfusions. The spinal fluid gradually cleared, the first sterile culture being reported on May 17, eight days after operation. On May 22 culture of the spinal fluid was positive again, but thereafter it was negative. Clinically, the child showed marked improvement; the meningeal signs disappeared in five days. The dural slits remained open for one week. On June 12 the child was discharged well.

The data on the spinal fluid throughout the illness were as follows:

Date, 1937	Culture	Cell Count
April 29	Positive, Pneumococcus type IV	4,280
May 1	Positive, Pneumococcus type IV	4,100
May 3	Positive, Pneumococcus type IV	3,000
May 5	Positive, Pneumococcus type IV	1,300
May 7	Positive, Pneumococcus type IV	4,000
May 11	Positive, Pneumococcus type IV	2,630
May 13	Positive, Pneumococcus type IV	620
May 17	Sterile	860
May 19	Sterile	70
May 21	Sterile	120
May 22	Positive, Pneumococcus type IV	600
May 23	Sterile	840
May 24	Sterile	430
May 25	Sterile	78
May 29	Sterile	40
June 3	Sterile	12

This is the third case in which sulfanilamide in itself was not sufficient to effect a cure and to bring about the ultimate result; it was again necessary to resort to decompression and slitting of the dura.

CASE 7.—L. F., a 22 year old white man, was admitted to the hospital on April 13, 1937, for submucous resection. He had had nasal obstruction as long as he could remember, present on both sides but more pronounced on the left. He used one handkerchief daily; his sense of smell was keen, and he did not suffer from headaches. His past history was irrelevant.

General physical examination gave negative results. Examination of the nose revealed a sigmoid deviation, first to the left and then to the right, in the region of the perpendicular plate of the ethmoid sinus, impinging on the left middle turbinate. The airways were poor bilaterally, that on the left being limited to a small passageway along the floor. The mucosa was reddened and thickened and did not shrink well when sprayed with a 4 per cent solution of cocaine. Both anterior and middle tips were hypertrophied and somewhat polypoid. Frank pus and polypi were not present. The nasopharynx was normal. The throat contained small tonsillar tabs left after enucleation. The ears and the larynx were normal. Transillumination was easily accomplished. The sinal plates were normal. Diagnoses of deviated septum and hypertrophic rhinitis were made.

On April 13 submucous resection was performed by one of the house surgeons. The anesthetic was a 10 per cent solution of cocaine sprayed into the nose under 25 pounds (11 Kg.) of pressure. The nose had first been washed with compound solution of sodium borate and the vibrissae cut. The usual Killian incision was made on the left and the resection performed without unusual difficulty or mishap except for a small perforation in the right flap about halfway back. The cartilage and bone were removed back to the rostrum of the sphenoid and down to the floor. The anterior tip on the left side was then removed with scissors and snare. On the right side the nasal snare broke so that a new wire could not be inserted. The procedure was completed with the use of a tonsil snare. The nose was packed with petrolatum gauze.

The next day the patient vomited, and the temperature rose to 105 F. The packing was removed. The patient complained of severe frontal headache, and a spinal tap was performed. This revealed cloudy fluid under a pressure of 150 mm. of water. A smear showed 1,400 cells, with 85 per cent polymorphonuclears. No organisms were visible on smear. Culture showed Pneumococcus

type XXIX. There was slight reduction with Benedict's solution. The sugar content was 61.7 mg. per hundred cubic centimeters and the chloride content 756 mg. Sulfanilamide and neoprontosil therapy was immediately initiated, one ampule of neoprontosil each six hours, 3 tablets (15 grains [1 Gm.]) of sulfanilamide each four hours being given. The patient received three blood transfusions of 500 cc. each. Daily spinal taps were done, and the spinal fluid was replaced with a solution of 0.8 Gm. of sulfanilamide crystals in 100 cc. of physiologic solution of sodium chloride.

Clinically, the patient made an almost immediate response; his temperature dropped, and four days later it was normal. His headache rapidly cleared, and in a week he was free of meningeal signs. Spinal fluid cultures were positive until eight days after operation. Two weeks after operation the cell count was 9 per cubic millimeter, and reduction with Benedict's solution was normal. Administration of sulfanilamide was continued, however, 10 grains (0.6 Gm.), and then 5 grains (0.3 Gm.), each four hours, until the patient's discharge from the hospital on May 19.

The data on the spinal fluid throughout the illness were as follows:

Date, 1937	Culture	Cell Count
April 14	Positive, Pneumococcus type XXIX	1,400
April 15	Positive, Pneumococcus type XXIX	4,000
April 16	Positive, Pneumococcus type XXIX	3,800
April 17	Positive, Pneumococcus type XXIX	4,000
April 18	Positive, Pneumococcus type XXIX	7,776
April 19	Positive, Pneumococcus type XXIX	544
April 20	Positive, Pneumococcus type XXIX	3,240
April 21	Positive, Pneumococcus type XXIX	1,800
April 22	Sterile	260
April 23	Sterile	114
April 24	Sterile	180
April 26	Sterile	28
May 15	Sterile	3

An attempt at surgical decompression in this case seemed out of the question, as a proper point of decompression could not be ascertained. Consequently, it seemed necessary to rely entirely on such measures as administration of sulfanilamide, spinal taps and transfusions. This is the only case in this series in which decompression and dural slitting was not used.

CASE 8.—D. M., a 9½ year old white boy, was admitted to the hospital on April 18, 1937, with a three days' history of earache on the right and full-blown meningeal signs. The ears revealed slight abnormality; Shrapnell's membrane was slightly injected on the right side. All landmarks were present. Because of the toxic, irrational state of the patient, the presence of tenderness over the mastoid was difficult to determine. The nose appeared clean. It was reported from Dr. Neal's laboratory that the spinal fluid contained Pneumococcus type I.

Mastoidectomy on the right side was done immediately. Extensive necrosis and softening of the right side was discovered, with pathologic exposures of the dura and sinus. Temporal decompression was done, and dural slits were made. Cultures of material from the mastoid showed Pneumococcus type I. Intensive therapy with antipneumococcus serum, neoprontosil and sulfanilamide was initiated, and several blood transfusions were given. In spite of these measures, the child rapidly declined, surviving for a week and dying on April 25, one week after admission to the hospital.

The data on the spinal fluid throughout the illness were as follows:

Date, 1937	Culture	Cell Count
April 18	Positive, Pneumococcus type I	2,484
April 19	Positive, Pneumococcus type I	1,764
April 20	Positive, Pneumococcus type I	4,320
April 21	Positive, Pneumococcus type I	7,200
April 22	Positive, Pneumococcus type I	4,752
April 23	Positive, Pneumococcus type I	8,910
April 24	Positive, Pneumococcus type I	3,024
April 25	Positive, Pneumococcus type I	3,132

CASE 9.—D. C., a 4 year old white boy, was admitted to Queensboro Pavillion on April 18, 1937, with a diagnosis of measles and a discharge from the left ear. Examination showed the discharge from the left ear and tenderness over the mastoid, as well as indications of catarrhal disease of the right middle ear. During the patient's stay at Queensboro, his temperature decreased gradually from 105 F. to 101. During this period he suffered an attack of bilateral acute ethmoiditis and swelling of the eyelids. The left ear continued to discharge, and the area over the left mastoid remained tender. Myringotomy was done on the right, and only a small amount of thin seropurulent discharge was obtained. On April 30 the boy was transferred to Queens General Hospital. The left ear now showed sagging of the wall of the canal and postauricular edema; the right ear had a mucopurulent discharge but no evidence of involvement of the mastoid.

On May 1 simple mastoidectomy was done on the left, with the finding of marked destruction in a large, well pneumatized mastoid. There were no pathologic exposures. On May 2, paresis of the left external rectus muscle was noted, and petrositis was suspected. On May 3, swelling was visible over the left anterior temporal region, but this subsided under wet dressings. The following day the temperature rose to 104 F.; nuchal stiffness was present, and other signs of meningitic involvement were noticed. Spinal tap showed cloudy fluid, with a cell count of 4,000, which yielded hemolytic streptococci on culture.

On May 12, simple mastoidectomy was done on the right, and extensive destruction was found in the region of the solid angle, as well as exposure of the bulbar end of the sinus. On account of these findings and the meningeal manifestations, temporal decompression and incisions of the dura were added to the operation. The foot of the bed was elevated, and administration of sulfanilamide, neoprontosil and sulfanilamide crystals intraspinally was begun immediately. Culture of the spinal fluid was positive on nine consecutive days, until May 13, when it was sterile, and the patient improved steadily. On May 24 the spinal fluid was clear, with no globulin and about 180 cells. The mastoidectomy wounds healed cleanly, and only the left middle ear showed a slight discharge on the patient's return home. At the time of his discharge from the hospital, on June 14, the spinal fluid had been normal for one month.

The data on the spinal fluid throughout the illness were as follows:

Date, 1937	Culture	Cell Count
May 4	Positive, Str. haemolyticus	4,000
May 5	Positive, Str. haemolyticus	3,150
May 6	Positive, Str. haemolyticus	378
May 6	Positive, Str. haemolyticus	2,160
May 8	Positive, Str. haemolyticus	390
Bay 9	Positive, Str. haemolyticus	2,140
May 10	Positive, Str. haemolyticus	1,160
May 11	Positive, Str. haemolyticus	940
May 13	Sterile	150
May 14	Sterile	640
May 16	Sterile	180
May 19	Sterile	60
May 24	Sterile	180
May 28	Sterile	12

This case is similar to cases 1, 2 and 4 in that all 4 were cases of invasion by hemolytic streptococci from which the patient ultimately recovered.

CASE 10.—E. H. was admitted to the hospital on May 1, 1938. At the time of admission the boy's right ear had been draining for six days. Three days prior to admission the temperature rose, and stiffness of the neck developed, accompanied by vomiting.

Physical examination revealed desquamation of the skin on the toes and fingers, postnasal discharge, discharge of thick pus from the right ear, nuchal and dorsal rigidity and bilateral Kernig and Babinski signs. The patient had received 40 grains (2.6 Gm.), 20 grains (1.3 Gm.) and 10 grains (0.6 Gm.) of sulfanilamide on the three days prior to admission. A spinal tap on admission showed cloudy fluid under increased pressure, with 2,800 cells. The dextrose content was reduced and the protein content increased. Seventy per cent of the cells were polymorphonuclears. Organisms were not seen on smear.

On May 3 mastoidectomy was done. Some necrotic bone was found. The dura was incised in two places; the wound was left open. One hundred and fifty cubic centimeters of citrated blood was transfused. The patient received sulfanilamide by mouth and neoprontosil and crystalline sulfanilamide intraspinally. Spinal taps were done daily. Culture of the spinal fluid taken on May 5 showed Str. nonhaemolyticus; the fluid contained 860 cells. On May 6 the spinal fluid contained Str. nonhaemolyticus, with 1,000 cells. Successive spinal taps showed continuous improvement.

The patient improved steadily, with no exacerbations. On June 23 he was discharged, with the ears dry and the mastoid healed. Residual neurologic abnormalities were not present.

A blood culture taken on May 2, 1938, was sterile. Culture of pus from the canal of the right ear on May 1, 1938, showed Staphylococcus aureus haemolyticus.

Sulfanilamide was given as follows: 20 grains (1.3 Gm.) on admission, 5 grains (0.3 Gm.) each three hours, eight doses in twenty-four hours, for seven days, then 5 grains each four hours for one day and then three times a day, and 15 cc. of an 8 per cent solution of crystalline sulfanilamide, intraspinally on May 3 and May 8. Transfusions of 150 cc. of citrated blood were given on May 3 and May 14, 1938.

The patient was discharged well on June 23. The data on the spinal fluid throughout the illness were as follows:

Date, 1938	Culture	Cell Count
May 5	Positive. Str. nonhaemolyticus	860
May 6	Positive, Str. nonhaemolyticus	1,000
May 7	Sterile	30
May 8	Sterile	

CASE 11.—A. M. K., a girl aged 8 years, was admitted to the hospital on May 10, 1938. The child had a cold two weeks prior to admission; three days before, the left ear began to discharge. The neck then became stiff. On the day of admission delirium and disorientation ensued, accompanied by vomiting.

Physical examination revealed an acutely ill child, with a moderately thick discharge from the left ear, redness and fulness of the drum, no sagging of the canal wall, apparent tenderness over the tip of the mastoid and stiffness in the neck.

A spinal tap showed cloudy fluid, with 2,500 cells per cubic centimeter, no sugar and a 4 plus reaction for globulin.

On May 10 the patient was taken to the operating room; necrotic cells were found in the zygoma.

The dura over the antrum was pathologically exposed and necrosis and free pus were found in the tip. A temporal decompression was done. On May 12 spinal fluid was reported to contain Str. haemolyticus. The child was taken to the operating room again, and dural slits were made, with escape of cloudy spinal fluid.

The child was subjected to transfusions, daily spinal taps and administration of sulfanilamide and neoprontosil, the latter intramuscularly and intraspinally.

On May 17 the temperature came down to normal, and it remained so. On June 3 plastic closure of the postauricular wound was done. Recovery was uneventful. The patient was discharged on June 6 to the outpatient department, which she attended from June 8 to June 25, when she was discharged, with the postauricular wound healed and the middle ear dry.

The data on the spinal fluid throughout the illness were as follows:

Date, 1938	Culture	Cell Count
May 12	Positive, Str. haemolyticus	2,300
May 13	Positive, Str. haemolyticus	1,450
May 14	Sterile	240
May 16	Sterile	30

CASE 12.—D. T., a man aged 41, was admitted to the hospital on March 3, 1938, with a history of a cold, beginning two weeks prior to admission and followed in a few days by earache on the left. Eight days prior to admission the ear began to discharge. On the night before admission severe headache ensued. On the morning of admission the patient refused food, vomited and showed signs of irritability. Convulsions or coma were not present.

Physical examination revealed an acutely ill patient, complaining of left hemicrania, with exquisite tenderness over the mastoid. The left ear had a full drum and a scant, thin discharge through a central perforation, without sagging of the wall of the canal. Mild nuchal rigidity was present; other observations were negative. A spinal tap revealed cloudy fluid under pressure with 800 cells per cubic millimeter, without sugar and with a 1 plus reaction for globulin. Organisms were not seen on smear.

On March 3 mastoidectomy was done on the left. Necrotic cells filled with pus, found in the tip and deep under the horizontal canal, were cleaned out. Pathologic exposure of the sinus, which was enlarged, and of the dura was present. After operation the patient received infusions and sulfanilamide therapy. The mastoid pus contained Str. haemolyticus. A culture of the spinal fluid was sterile. The patient did well for seven days, with the spinal fluid clearing, and then complained of headache again. A spinal tap revealed 2,200 cells per cubic millimeter. The fluid cleared rapidly, however, with repeated taps and administration of sulfanilamide, so that the patient was discharged on March 18 to the outpatient department for dressings.

He attended the outpatient department from March 22 to April 2. Good progress was noted; granulations were forming in the mastoidectomy wound. The middle ear continued to drain however.

On April 6 the patient was admitted again; he complained of frontal headaches and fever of two weeks' duration.

Physical examination revealed a thick pulsating discharge from the left ear and profuse drainage from the mastoidectomy wound. In spite of the absence of meningeal signs, a spinal tap was done, revealing 450 cells per cubic millimeter and Str. haemolyticus on culture.

On April 7 the mastoidectomy was revised and the dura slit for drainage. No focus of infection was found; the sinus was inadvertently opened and was packed off. The patient continued to do badly. He was comatose, with stiffness of the neck and a temperature of 103 F. On April 10 the operation was repeated; a lead was found under the facial canal, going in the direction of the petrous tip. It was cleaned out and enlarged, and more dural slits were made. Culture of the spinal fluid yielded Str. haemolyticus; the cell count was 1,650. The patient was given blood transfusions.

Owing to the increased cell count of the spinal fluid and the increasing coma of the patient, another exploration was made, on April 11, for an abscess. A good-sized abscess was found in the left temporal lobe, 6 or 7 cc. of pus evacuated and a Mosher drain inserted.

The patient died the next day, April 12.

The data on the spinal fluid throughout the illness were as follows:

Date, 1938	Culture	Cell Count
March 3	Sterile	800
March 10	Sterile	2,200
April 6	Positive, Str. haemolyticus	450
April 10	Positive, Str. haemolyticus	1,650

The patient apparently had an abscess of the temporal lobe from the beginning, which was not discovered until it was too late.

CASE 13.—F. K., a married man aged 33, had as his chief complaint the results of an accident on Sept. 5, 1938, when the automobile in which he was riding was hit by a truck. As a result of the collision, the patient was thrown against the door and hit his head against it. He recalled nothing from then on. His relatives stated that there was bleeding from the ears, more so from the right one. He vomited several times. On admission to the hospital one day later, after being transferred from Trinity Hospital, he was drowsy but conscious.

Physical examination revealed a drowsy patient, answering questions with difficulty, slow but conscious. Nuchal rigidity and a serosanguineous discharge from the right ear were marked. The skin on the right side of the face could not be wrinkled; the right half of the mouth also was paralyzed. The tongue was coated and deviated toward the left side. The reflexes and the Babinski sign were absent. The pupils reacted to light and were equal. The opinion was that cerebral contusion was present, with moderate subdural hemorrhage (left motor region) and fracture of the skull. The roentgen report on the skull was as follows: "Examination of the skull shows a long linear fracture on the right side. It extends from the squamous portion of the temporal bone, across the temporoparietal suture and directly upward to the vertex. There is another fracture, extending from the region of the foregoing fracture, across the temporosphenoid area and into the anterior portion of the parietal bone. The fracture lines are approximately 5 and 2 inches (13 and 6 cm.) in length, respectively. There is no depression or marked separation of the fragments (Sept. 7, 1938)."

A study of the cervical part of the spine did not show evidence of fracture or dislocation. In passing, it was noted that there was a fracture involving the zygomatic arch on the right side.

On October 5 mastoidectomy with decompression of the temporal bone was done on the right. As the skin was retracted it was noted that there was a fracture extending from the zygoma up about 1 inch (2.5 cm.), then across, over the squamous portion of the temporal bone, for another inch (2.5 cm.) and then horizontally for 1½ inch (4 cm.).

After the cortex was removed, it was noted that there were spicules of bone going into the dura along the line of fracture.

The cells of the initial groove were gouged out, and as the operation proceeded toward the zygoma marked destruction was noted, and immediately a large-sized cavity was entered, which had exposed the dura. The cells of the zygoma were thoroughly cleaned out, and the semicircular horizontal canal presented itself; above and below it there seemed to be destruction of cells; these were cleaned out. The antrum was thoroughly cleaned out. Then the squamous portion of the temporal bone, from the zygoma up and around for about 2½ inches (6 cm.), was removed, and it was seen that the dura was dark, with some exudate. The squamous portion of the temporal bone was removed until healthy, normal dura was found. The cells over the sinal plate were cleaned; the dural plate was removed for at least 1 inch (2.5 cm), and the tip was cleaned. A 95 per cent solution of alcohol was then put into the wound for several minutes. Gloves and gown were changed, and a new knife was used to nick the dura, horizontally, for about ½ inch (1.3 cm.). The wound was left open and dressings applied.

The patient was discharged on December 23. He was seen on December 30; the paralysis of the facial nerve was clearing up, and the wound closing over. On Jan. 1, 5, 10, 15 and 30, 1939, the general condition was good; the wound was practically healed, except for a small opening. On February 15 the wound still had an opening, about the size of a pea; the paralysis of the facial nerve was clearing up. On March 3 the wound was closed and the paralysis of the facial nerve was better.

The data on the spinal fluid throughout the illness were as follows:

Date, 1939	Culture	Cell Count
September 10	Positive, Staph. albus	3,850
September 12	Positive, Staph. albus	2,860
September 16	Sterile	1,420
October 9	Sterile	600
October 14	Sterile	240
October 20	Sterile	150

CASE 14.—A. G., a boy aged 5½ years, was admitted to the hospital on Jan. 29, 1939. Two nights before admission the patient complained of pain in the right ear. The temperature was 102 F. The next morning the pain in the ear was worse, and at night the temperature was 106 F., with nuchal rigidity, and the child was admitted to the hospital.

Physical examination showed the right ear drum at the time of admission red and bulging. Landmarks were not visible.

Myringotomy was done, and a mixture of blood and pus was obtained. A culture of this discharge contained Staphylococcus aureus and B. influenzae. The left drum was catarrhal. Full-blown signs of meningitis were present.

A spinal tap revealed fluid under increased pressure; 40 cc. of cloudy fluid was removed; it contained 4,840 cells and yielded gram-positive bacilli on smear. Culture showed B. influenzae. Roentgen examination of the mastoid revealed diffuse haziness throughout the right side. No destruction of cellular walls was noted.

Mastoidectomy was then done on the right. The mucous membrane throughout was extremely hemorrhagic; frank pus was not encountered; the cells in the zygoma were softened. On the day of admission, the patient received 20 grains (1.3 Gm.) of sulfanilamide, with the dose repeated in one hour, and then 10 grains (0.6 Gm.) every four hours.

Postoperatively the patient was subjected to two spinal taps daily, with anti-influenza serum given intrathecally at each tap (eight times in all). Twenty grains (1.3 Gm.) of sulfapyridine and 10 grains (0.6 Gm.) of sodium bicarbonate were then given every four hours, and continuous intravenous administration of a 10 per cent solution of dextrose in a saline solution was carried out. The patient received daily small transfusions. The temperature on the day of operation varied between 103 and 105 F.; the next day it fell from 105 to 99, but on the next day it returned to 105. The patient was stuporous; his condition was poor, and he was jaundiced. Postoperatively he was kept in the Trendelenburg position, and the flow of spinal fluid from the mastoidectomy wound was fairly copious. The drainage persisted freely for about forty-eight hours. The temperature on the day after operation varied between 103 and 105 F. but on the next day started to decline; the patient was slightly more alert. The temperature subsided in the next four days to normal, at which it remained; the cell count of the spinal fluid dropped consistently and steadily. Recovery thereafter was uneventful.

Administration of sulfapyridine in the original dose was continued until February 17, when the dose was cut to 7½ grains (0.5 Gm.) three times daily. On February 27 all medication was stopped.

On March 1 the child was discharged. He has returned for observation at weekly intervals and looks well at the time of writing. The wound was practically closed without evidence of herniation or discharge on March 14.

The data on the spinal fluid throughout the illness were as follows:

Date, 1939	Culture	Cell Count
January 29	Positive, B. influenzae	4,840
January 30	Positive, B. influenzae	3,650
January 31	Positive, B. influenzae	2,100
February 2	Positive, B. influenzae	2,350
February 3	Positive, B. influenzae	1,960
February 5	Positive, B. influenzae	2,260
February 9	Sterile	1,350
February 12	Sterile	680
February 14	Sterile	920
February 16	Sterile	50

CASE 15.—V. S., a girl aged 8, was admitted to the hospital on Feb. 14, 1939, with a history of earache on the left for seven days and a purulent discharge for three days, following acute infection of the upper part of the respiratory tract. Some tenderness was present over the left mastoid. Simple mastoidectomy was done on the left on February 25, because of the tenderness over the mastoid and the purulent aural discharge, with a temperature of 100 to 104 F. and roentgen signs of mastoiditis.

The operative findings were reported as pus welling up on the making of the initial groove, pneumatic and extensive cells and pus in the retrosinal and zygomatic cells. Culture of material from the mastoid revealed Str. haemolyticus. The postoperative course was uneventful. The patient was discharged on March 6 to the outpatient department, where the wound was dressed.

Because of a fistula at the upper angle of the wound, the child was readmitted on March 25. On March 27 the mastoidectomy was revised; a few cells in the

zygoma and the tip were cleaned out. Profuse drainage, frontal headache on the left, diplopia and palsy of the left external rectus muscle were noted. Petrositis on the left was reported as shown on a roentgenogram taken on April 7. The temperature was 102 or 103 F. A spinal tap done on April 8 showed cloudy fluid under 200 mm. of pressure, with 3,200 cells (mainly polymorphonuclears) per cubic millimeter, which yielded a positive culture for Str. haemolyticus. Organisms were not seen on smear; stiffness of the neck or a positive Kernig sign was not present.

On April 8 a third operation, revision of the mastoidectomy, exposure of the dura and modified radical mastoidectomy, was done. The dural plate of the middle fossa was soft; the dura looked fairly normal. A small area of soft necrotic bone posterior to the horizontal semicircular canal was found and curetted toward the petrous apex. The soft bone extended inward about 0.5 cm. The postoperative condition was poor. The patient received 250 cc. of blood and 600 cc. of a 5 per cent solution of dextrose in a saline solution. On March 4 the patient began to receive sulfanilamide (with sodium bicarbonate), 45 grains (2.9 Gm.) and then 10 grains (0.6 Gm.) every four hours. A spinal tap on March 9 showed 120 mm. of pressure and a cell count of 1,100 per cubic millimeter and yielded a culture containing Str. haemolyticus. Another tap showed 900 cells, mostly polymorphonuclears.

On April 10 radical mastoidectomy was done, with almost complete removal of the tegmen of the mastoid and middle ear; a large cavity was made into the petrous pyramid below the canals, and the canals were skeletonized. A culture of the spinal fluid contained Str. haemolyticus on two occasions. Organisms were not seen on smear; the fluid did not contain sugar; the protein content was increased.

Treatment, in addition to the operations, consisted of administration of sulfanilamide (with sodium bicarbonate), daily blood transfusions and daily spinal taps. On April 9, 1939, the dose of sulfanilamide was 45 grains (2.9 Gm.) and then 10 grains (0.6 Gm.) every four hours; on April 10, 95 grains (6.2 Gm.) and then 20 grains (1.3 Gm.) every four hours, and for eight days subsequently, the same (120 grains [7.8 Gm.] daily), with 5 cc. of neoprontosil every four hours added on April 14. On April 18 the dose was cut to 5 grains (0.3 Gm.) every four hours, which the patient was receiving on April 21.

Two hundred cubic centimeters of blood were given on April 8 and April 9, 1939, and then 75 cc. daily.

The temperature is normal at the time of writing, and the child looks fairly well and has no complaints. The spinal fluid was sterile on April 19, with 350 cells, and she seemed on the road to recovery.

On April 25 she was out of bed and doing nicely.

On May 10 she was discharged well.

The data on the spinal fluid throughout the illness were as follows:

Date, 1939	Culture	Cell Count
April 8	Positive, Str. haemolyticus	3,200
April 9	Positive, Str. haemolyticus	1,100
April 10	Positive, Str. haemolyticus	10,000
April 11	Positive, Str. haemolyticus	14,000
April 19	Sterile	350
April 22	Sterile	120

CONCLUSIONS

In this series of 15 consecutive cases of meningitis, 11 patients recovered and 4 died, giving a mortality of 27 per cent.

Sulfanilamide, while obviously extremely important, is apparently not sufficient in itself to produce a cure.

Surgical intervention in itself is not sufficient, and it seems that a combination of the two, namely, sulfanilamide and surgical intervention offers the best hope.

In the use of the drug, it is important that treatment be started early, that the dose be large and that the treatment be continued for several weeks after cessation of symptoms.

Surgical measures likewise should be instituted as soon as the diagnosis is made.

In 7 of 8 cases in which the invading organism was Str. haemolyticus the patient recovered. In 2 cases in which the organism was Pneumococcus type I and 1 case in which the organism was Pneumococcus type III the patient died.

Had sulfapyridine been known when the pneumococcic infections were treated, the results might have been better.

Six patients showed paralysis of the external rectus muscle.

While I realize that this is not a large series of cases, it seems, nevertheless, that with the use of sulfanilamide plus surgical intervention meningitis is not necessarily to be considered so formidable as formerly.

My associate, Dr. A. A. Cirillo, cooperated in the handling of the cases, and the residents, Dr. P. Lefkowitz and Dr. E. M. Hipsch, worked up the series.

ABSTRACT OF DISCUSSION

DR. WELLS P. EAGLETON, Newark, N. J.: I wish to call attention to certain facts about the cure of meningitis that have long been known but apparently today are being disregarded.

I disagree with Dr. Cunning as to meningitis having been always fatal, for I would point out that before we had sulfanilamide, I cured 27 patients with bacteria in the spinal fluid. With all cases of meningitis taken together, my percentage of recoveries was 32.

Examination of patients with bacteria in the fluid who were cured without the use of sulfanilamide taught certain facts that help to explain why patients are cured today when treated with sulfanilamide and why certain of them relapse.

All the patients who recovered were cured by removal of all foci of infection in the bone or vessel and because the bacteria in the spinal fluid were attenuated, of low grade virulence or few. Contributing factors were that the sugar content of the cerebrospinal fluid was maintained, although the patients often were acutely ill, and that the infection had lasted a sufficient time to allow protective meningitis to develop. Several of the infections which I cured belonged to the specific self-limiting type, which I have named toxemic and allergic overflow meningitis from scarlet fever or from pneumonia.

The presence of sugar in the cerebrospinal fluid signified that the normal metabolic action of the brain had not been interfered with; although the meninges were the seat of a bacterial infection, the metabolic action of the central mechanism continued. This is exactly what takes place from the use of sulfanilamide. The drug acts in a similar way, by causing bacteriostasis, thus

preventing the bacteria from paralyzing the metabolic mechanism of the brain. The relapses occur because sulfanilamide has a bacteriostatic action only on the bacteria that it can reach.

The case of death from abscess of the brain just reported is enlightening. It reminds me of one of my cases, in which a child apparently suffered from meningitis. A radical operation on the bone was done. He was given sulfanilamide. The spinal fluid rapidly cleared of micro-organisms, but the child did not get well.

A ventriculogram revealed the abscess in the occipital lobe, which was opened. The pus was full of streptococci, and yet the cerebrospinal fluid had been free from micro-organisms for many weeks, during which time the patient was given sulfanilamide continuously.

The presence of hemolytic streptococci in the abscess of the brain after sulfanilamide had sterilized the cerebrospinal fluid meant that sulfanilamide does not act in the presence of encapsulated or necrotic tissue even when in high concentration in the blood or cerebrospinal fluid. It cannot reach a localized area of infection or attack bacteria in areas of necrotic tissue.

Sulfanilamide acts best in the presence of fever, and in meningitis there is fever, but with abscess of the brain the temperature often is subnormal.

I would emphasize, as has Dr. Cunning, that, while this drug is a most wonderful remedy, one must be guided by the old surgical principles. In every case of otitic meningitis one must thoroughly explore and remove every possible focus of infection. A clinical diagnosis of the exact anatomic location of the foci of infection in the bone or vessels can be made in the vast majority of cases if a proper history is taken and can invariably be made when this study of the case is followed by complete surgical exposure of all possible tracks of infection from the bone into the meninges. This is possible only by a complete unlocking operation. To obtain the best results all localized foci of infection in or around the temporal bone must be drained, and if possible eradicated. The only satisfactory operative technic ever devised that fully exposes all areas of infection is what I call the "unlocking of the petrous pyramid."

The carotid artery should always be tied when the infection in the meninges comes from, or the advancing lesion is, thrombophlebitis of the carotid venous plexus.

The dura should be opened when it contains a localized focus adjacent to the primary focus in the bone, as in the vast majority of cases of otitic meningitis.

Dr. Bernard J. McMahon, St. Louis: This interesting and carefully prepared paper has presented for consideration many significant points in the treatment of otitic infections and their complications.

Dr. Cunning has brought out the important point, that chemotherapy, serotherapy, transfusion and other adjunct treatment should be supplemented by the proper surgical intervention, which should not be inadvisedly postponed in the hope that the adjunct measures may accomplish the desired recovery.

Incisions of the meninges establish drainage not only from the intracranial cavity but from the meninges themselves. The meningeal drainage is important in releasing the inflammatory exudate in these membranes themselves about the perivascular lymphatics along which the infection has extended from the bone of the mastoid to the cranial cavity.

The indications for paracentesis or mastoidectomy remain unchanged.

The use of sulfanilamide will not and cannot obviate operative intervention in the presence of necrosis or cavitation within the cellular structure of the

mastoid process. The presence of the hemolytic streptococcus should be positively ascertained if possible before this medication in larger doses is started, and the dosage should be guided by the severity of the infection and subsequently adjusted according to the reaction of the patient. One should always bear in mind the numerous signs of sulfanilamide toxicity which have been repeatedly reported. The onset of these signs is not always in direct proportion to the dose, as many persons are particularly susceptible to the drug.

Dr. Converse has mentioned the importance of hemotherapy or serotherapy when the response to sulfanilamide therapy is inadequate, since without the timely mobilization of the phagocytic cells at the area of infection chemotherapy will be unavailing. Sulfanilamide can exert a bacteriostatic effect for only a certain length of time before toxic signs may develop; consequently, the adjunct treatments should be resorted to within the first forty-eight to seventy-two hours.

Sulfanilamide should not be administered unless the patient is kept in bed and under the careful and constant observation of his physician.

It is the duty of the physician to warn patients of the dangers of ill advised and uncontrolled administration of this drug, and one may hopefully look forward to the enactment of legislation in every state prohibiting the sale of sulfanilamide, sulfapyridine and their allied products by the druggist without a physician's prescription.

Dr. A. A. Cirillo, New York: I should like to stress 1 particular case in Dr. Cunning's series, which was observed recently at Queens General Hospital. A child 5 years of age was admitted on January 29 with influenzal meningitis. Culture and smear were positive. Simple mastoidectomy was performed and sulfapyridine, serum and sulfanilamide were administered. Because the child was getting worse Dr. Cunning suggested that a dural slit be made. The child had a temperature of 106 F. and a pulse rate of 136 and was jaundiced and comatose.

Two days later the temperature dropped to 98 F., and on March 3 the child was discharged as cured.

In a case of meningitis due to Staph. albus resulting from an automobile accident paralysis of the right facial nerve and hemorrhage from the right ear developed. Staph. albus was present in the spinal fluid and could be demonstrated on smear. It was noted in doing a decompression in this case that two spicules of bone had perforated the dura; this was evidently the avenue of infection. This patient, too, recovered.

Dr. Pierre Viole, Los Angeles: Apropos of the remarks of the essayist concerning the use of serum, I should like to stress its importance, particularly in the presence of infection with hemolytic streptococci. In Los Angeles some spectacular results have been obtained in such cases with the use of human convalescent scarlet fever serum obtained from the serum center at the Children's Hospital there. In view of the successes with this treatment, I think its use should be encouraged. I should be interested also in hearing reports from other medical centers where human convalescent serum has been used extensively.

INTRANASAL DACRYOCYSTOTOMY

W. E. SAUER, M.D.
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Surgery of the lacrimal sac dates back to the time of Galen, 50 A.D., and of Celsus,[1] 100 A.D., but credit for the first intranasal dacryocystotomy is generally given to Caldwell,[2] of New York, who published his work in 1893. Ritter [3] cited the work of Hopmann, who as early as 1885 brought forth the idea of operating on the lacrimal sac intranasally, using a sound passed from above as a guide. Killian [4] in 1889 reported his intranasal operation. He passed a Bowman probe from above, carrying it down into the lacrimal canal as far as possible. He then cut off the anterior end of the inferior turbinate and removed all the intranasal wall of the nasolacrimal duct. Passow [5] reported on 4 patients operated on by this method two years later. In 1904 Strazza reported his intranasal operation and in 1908 Okuneff reported on a similar operation. All these early workers removed a part of the inferior turbinate intranasally and then opened the nasolacrimal duct, working upward until a probe passed from above could easily be advanced into the nose. Only Strazza continued his resection upward to include the tear sac; he applied the curet to its walls.

However, it was West [6] who put the intranasal operation on a firm basis. His first publication appeared in 1909. It is interesting to follow the evolution of his technic. In his first case he followed Killian's method—i. e., the anterior part of the inferior concha was removed,

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1. Celsus, A. C.: Of Medicine, in Eight Books, translated by J. Greive, ed. 3, London, E. Portwine, 1837.

2. Caldwell, G. W.: Two New Operations for Obstruction of the Nasal Duct, with Preservation of the Canaliculi, and an Incidental Description of a New Lachrymal Probe, New York M. J. **57**:581, 1893; reprinted, Am. J. Ophth. **10**:189, 1893; A New Operation for the Radical Cure of Obstruction of the Nasal Duct, New York M. J. **58**:476, 1893.

3. Ritter, in Katz, L., and Blumenfeld, F.: Handbuch der speziellen Chirurgie des Ohres und der oberen Luftwege, Leipzig, Curt Kabitzsch, 1923.

4. Killian, cited by Ritter.[3]

5. Passow: Zur chirurgischen, Behandlung der Verengerungen des Thränen-Nasenkanals, München. med. Wchnschr. **48** (pt. 2):1403, 1901.

6. West, J. M.: A Window Resection of the Nasal Duct in Cases of Stenosis, Tr. Am. Ophth. Soc. **12**:654, 1909.

and the entire nasolacrimal duct was opened. Soon after that he [7] began to save the inferior concha; he opened the nasolacrimal duct by making a window in the lateral nasal wall above the inferior turbinate. A probe was passed into the nasolacrimal duct from above to serve as a guide. The next step in the development of his operation came when he opened only the sac; the duct was left completely alone. The final improvement came when he began to excise all the wall of the sac except a small piece immediately around the opening of the canaliculi—the so-called intranasal total extirpation. Briefly, he elevated a small flap of nasal mucoperiosteum over the torus lacrimalis and turned it down; then, by means of a chisel applied intranasally, he made an opening in the bone (including a part of the lacrimal bone and a part of the frontal process of the maxillary bone). Through this bony window he seized the wall of the sac (a probe having previously been passed in through the canaliculus to make tension on the wall) with a forceps and extirpated a part or all of it.

Polyák [8] and Halle [9] described a similar intranasal technic at about the same time. Polyák elevated a piece of nasal mucoperiosteum but did not save it. He made a wide exposure of the sac and then removed its entire nasal wall. It is interesting to recall that in 1902 Polyák [10] had published a method of retrograde dilation of the nasolacrimal duct. He later abandoned this. Halle in describing his operation laid great stress on his flap of nasal mucoperiosteum, which he turned back. After the bony window was made and the sac opened, he completely severed the sac from the duct; the flap was returned to its original position and a small hole cut out of it to serve as a passageway for the tears. The remainder of the flap formed a sort of valve to prevent the blowing of air up into the conjunctival sac from the nose.

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7. West, J. M.: Tränenträufeln, Deutsche med. Wchnschr. **38**: (pt. 1) 779, 1912; Die totale Exstirpation des Tränensackes von der Nase aus mit Wiederherstellung des normalen Abflusses in Fällen von Dakryozystitis, Ztschr. f. Augenh. **45**: 159, 1921; Intranasal Lacrimal Sac Operation, Arch. Ophth. **55**:351 (July) 1926.

8. Polyák, L.: Ueber das Eröffnen des Ductus nasolacrymalis im vorderen Teile des mittleren Nasenganges, Ztschr. f. Augenh. **27**:92, 1912; Ueber die intranasale Dacryocystotomie, ibid. **30**:557, 1913; Zwei Fälle Schussverletzungen der Nase, mit Verletzung des Tränensackes und Nebenhöhleneiterung, Internat. Centralbl. f. Laryng. **34**:67, 1918.

9. Halle: Zur intranasalen Operation am Tränensack, Arch. f. Laryng. u. Rhin. **28**:256, 1914; Internasale Tränensackoperation bei einen Säugling von 3½ Monaten zur Entfernung einer hineingeglitten Dauersonde. Die intranasale Tränensackoperation und ihre Erfolge, Berl. klin. Wchnschr. **55**:256, 1918; Die nasale Chirurgie der Tränenwege, Ztschr. f. Laryng. u. Rhin. **11**:60, 1923.

10. Polyák, L.: Die Sondierung des Ductus nasolacrymalis von der Nase aus, Arch. f. Laryng. u. Rhin. **12**:379, 1902.

From that time (about 1911) on, a wealth of material on intranasal operations was published; the numerous operative procedures embraced the same principle, but each had some peculiarity of technic. A few of them will be described briefly. Choronshitzky [11] passed a pointed metal sound through the punctum and canaliculus into the sac. He then forced this through the lacrimal bone into the nose and, using it as a guide, made a communication between the nasal cavity and the tear sac intranasally. Later he merely made a puncture through the lacrimal bone with the probe. He passed a thread through the tract and allowed it to remain for some time. In 1912 Dr. W. M. C. Bryan [12] reported a slight modification of West's operation. Glogau [13] made use of Halle's technic but left a continuous thread in place for several weeks. In 1915 Hanger [14] reported a technic essentially like Killian's. Rudolf Hoffmann [15] slit the upper canaliculus and through it passed a sound larger than could otherwise have been used, pushing the tip through the lacrimal bone into the nose to guide him in making an intranasal opening between the nasal cavity and the tear sac.

The idea of a thread or bougie remaining in an opening between the tear sac and the nose is an ancient one, and numerous ingenious methods have been devised to accomplish this. Sir Percival Pott [16] in the eighteenth century described an inlying leaden cannula and bougie; Andrew [17] in 1883 recommended an inlying leaden stylet; Vulpius [18] used an indwelling metal sound, leaving it in place for a year or more; Kyle [19] inserted a removable silver cannula, which was cleaned and replaced by the patient from time to time; Koster [20] used a silk thread;

11. Choronshitzky, B.: Die perkanalikuläre Tränensackdurchstechung als Einleitung zur intranasalen Tränensackeröffnung und als selbstandige Operation, Arch. f. Laryng. u. Rhin. **28**:363, 1914.

12. Bryan, W. M. C.: Submucous Dacryocystorhinostomy for Persistent Dacryocystitis, Ann. Ophth. **21**:497, 1912.

13. Glogau, O.: A Case of Dacryo-Cysto-Rhinostomy, Laryngoscope **25**:28, 1915.

14. Hanger, F. M.: An Intranasal Operation Without a Guide for the Cure of Dacryocystitis, Laryngoscope **25**:23, 1915.

15. Hoffmann, R.: Ueber Dakryocystorhinostomie, Monatschr. f. Ohrenh. **48**: 985, 1914.

16. Pott, P.: Of the Fistula Lachrymalis, in Chirurgical Works of Percival Pott, edited by J. Earle, first American edition, from the last London edition, Philadelphia, J. Webster, 1819, vol. 1, p. 179.

17. Andrew, E.: The Treatment of Lachrymal Obstruction, Brit. M. J. **2**: 1185, 1883.

18. Vulpius, W.: Die Behandlung der Thränennasencanalstenosen; eine rhinologische Aufgabe, Deutsche med. Wchnschr. **22**:99 (pt. 1), 1896.

19. Kyle, J. J.: Operation Intended as Substitute for Extirpation of Lachrymal Gland or Duct, Am. J. Ophth. **14**:369, 1897.

20. Koster, W.: Die permanente Drainage der Tränenabflusswege, Arch. f. Ophth. **67**:87, 1907.

Burdon-Cooper [21] used silk-worm gut; Van Lint [22] used rubber tubing, and as recently as 1936 Spratt,[23] of Minneapolis, recommended the use of a hollow metal tube.

Yankauer [24] suggested that the nasolacrimal duct acts like a siphon. Working intranasally, he elevated the nasal mucoperiosteum over the duct, resected its bony wall and slit the entire membranous canal from its nasal opening, up to and including the sac. Then he replaced the nasal mucoperiosteal layer. The result was an enlargement of the bony membranous passageway and the reestablishment of drainage in the natural manner. Naturally the anterior part of the inferior concha was sacrificed during this operation.

Horgan [25] always performed submucous resection of the septum before going ahead with intranasal dacryocystostomy (West type). Blegvad [26] did an intranasal operation similar to that of West, except that he used an electric drill rather than a chisel to go through the bone. In 1920 Bookwalter [27] reported an intranasal procedure essentially the same as West's. Affolter [28] made a window in the nasal septum, operating through it from the opposite nostril. This opening he closed after the operation. Good [29] reported a method similar to Yankauer's. Clark [30] published an account of his intranasal operation in 1922. He passed a probe through a small external incision into the sac rather than through a canaliculus. The probe was then forced on through the lacrimal bone and acted as a visible guide in the nose.

Numerous other methods of treatment for lacrimal obstruction and infection have been in use. In ancient times the tear sac was opened and a red hot cautery forced through the lacrimal bone into the nose.

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21. Burdon-Cooper, J.: A Silkworm-Gut Lachrymal Style, Ophth. Rev. **26**: 1, 1907.

22. Van Lint: Trepanation of the Os Unguis and Placing of a Rubber Drain in Chronic Dacryocystitis, Am. J. Ophth. **3**:367, 1920.

23. Spratt, C. N.: Use of Callahan Tubes in Treatment of Chronic Dacrocystitis, Am. J. Ophth. **19**:601, 1936.

24. Yankauer, S.: The Technic of Intranasal Operations upon the Lacrimal Apparatus, Laryngoscope **22**:1331, 1912.

25. Horgan, J. B.: The Operation of Dacryocystorhinostomy: Its Indications and After-Treatment, J. Laryng., Rhin. & Otol. **31**:225, 1916.

26. Blegvad, N. R.: Bemerkungen über die Westsche Operation, Klin. Monatsbl. f. Augenh. **65**:429, 1920.

27. Bookwalter, C. F.: Intranasal Dacryocystostomy, Arch. Ophth. **49**:568, (Nov.) 1920.

28. Affolter: Die temporäre Resektion der Nasenscheidewand bei intranasalen Tränensackoperation, Internat. Centralbl. f. Laryng. **35**:27, 1919.

29. Good, R. H.: Simplified Intranasal Operation for Obstruction of Nasolacrimal Duct, Am. J. Ophth. **4**:597, 1921.

30. Clark, J. S.: Combined Intra- and Extranasal Operation for Dacrocystitis, Illinois M. J. **42**:104, 1922.

Escharotics also were employed. Woolhouse and Platner in 1724 opened the sac externally and drained it by making a hole in the lacrimal bone into the nasal cavity. Berlin in 1868 advocated total extirpation of the tear sac. This idea was revived in 1903 by Axenfeld.[31] In 1904 Toti,[32] of Florence, brought out his external dacryocystorhinostomy, which has enjoyed wide popularity. Toti incised the skin and elevated the periosteum off the lacrimal bone and the frontal process of the maxillary bone in the lacrimal fossa. This layer of periosteum, plus the tear sac, was displaced laterally. The bony wall of the lacrimal fossa was resected. Windows were then made in the nasal mucoperichondrium and in the lacrimal sac to correspond to the bony window and to cover its raw edge. The external incision was closed. Lagrange and Aubaret[33] opened the sac by external incision, applied the curet to its walls and then made an opening into the nasal cavity for drainage. Dupuy-Dutemps and Bourguet[34] operated in essentially the same way as Toti, except that they actually sutured the cut edges of the wall of the sac and the nasal mucosa. This procedure is far superior, in that healing is immediate and no after treatment is required. Mosher[35] in 1921 and 1923 reported his modification of Toti's operation. Recently Chandler,[36] of Boston, compared a series of operations done by Mosher's technic, another by Dupuy-Dutemps' and Bourguet's technic and another by his own modification of the external operation. Stokes[37] made an external incision, freed the lacrimal sac and severed it from its lower end. This he implanted in a bony window, which he made through an opening in the nose. Kutvirt[38] described a complicated approach to the tear sac from

31. Axenfeld, T.: Die Exstirpation des Thränensackes zur Prophylaxe der septischen Infektion der Berufverletzungen des Auges, Klin. Monatsbl. f. Augenh. **41** (pt. 1):128, 1903.

32. Toti, A.: Zum Prinzip, zur Technik und zur Geschichte der Dakrocystorhinostomie, Ztschr. f. Augenh. **23**:232, 1910; Technique systematique de la dacryocystorhinostomie, Ann. d'ocul. **143**:417, 1910.

33. Lagrange and Aubaret: Contribution historique et clinique au traitement des dacryocystites par la création d'une voie nouvelle a l'unguis, Ann. d'ocul. **138**: 161, 1907.

34. Dupuy-Dutemps and Bourguet: Note préliminaire sur un procédé de dacryocystorhinostomie, Ann. d'ocul. **157**:445, 1920; Procédé plastique de dacryocystorhinostomie et ses résultats, ibid. **158**:241, 1921.

35. Mosher, H. P.: The Mosher-Toti Operation on the Lachrymal Sac, Laryngoscope **31**:284, 1921; The Combined Intranasal and External Operation on the Lacrimal Sac (Mosher-Toti), Ann. Otol., Rhin. & Laryng. **32**:1, 1923.

36. Chandler, P. A.: Dacryocystorhinostomy, Tr. Am. Ophth. Soc. **34**:240, 1936.

37. Stokes, W. H.: Transplantation (Implantation) of the Lacrimal Sac in Chronic Dacryocystitis, Tr. Am. Acad. Ophth. **43**:343, 1938.

38. Kutvirt, O.: Dacryorhinostomie par les voies orales, Rev. de laryng. **43**: 675, 1922.

the mouth; his method recalls a "Denker operation;" he resected the bony lateral nasal wall but did not open into the antrum. On the other hand, Von Eiken had previously reported draining the tear sac into the maxillary sinus.

Wiener and I [39] first reported our method in 1920 and I [40] again in 1923. Wood [41] in 1933 reported as follows: "This operation has gradually been perfected until it has become remarkably successful in properly selected cases." He stated that in cases in which there is any form of obstruction between the eye and the sac this operation is not suitable.

Our procedure is as follows:

About two hours before beginning the operation the patient is given ¼ grain (16 mg.) of morphine and $\frac{1}{130}$ grain (0.5 mg.) of scopolamine hydrobromide. A second dose of scopolamine hydrobromide may be required, but a second dose of morphine is never given.

A cotton plug soaked in a 0.5 per cent solution of pontocaine hydrochloride is pushed under the upper and lower lids at the inner canthus and permitted to stay for about two minutes. With a fine platinum needle of a small hypodermic syringe, the conjunctiva is punctured just below the lower punctum, and a few drops of a 2 per cent solution of procaine hydrochloride is injected; the needle is pushed forward along the border of the canaliculus, toward the sac, alternately to the conjunctival and the skin side, a small amount of the solution being injected each time. When the sac is reached, the needle is pushed toward the top of it, where a small amount is injected, and then toward the bottom of it, where a slightly larger amount is injected. This will catch all the terminal nerves supplying the canaliculus and the lacrimal sac, so that a probe can be passed without pain. The nose is cocainized in the usual way.

The punctum is dilated with a punctum dilator; the Galezowski dilator is then introduced, and the canaliculus is stretched. A Ziegler probe is passed into the punctum and along the canaliculus as far as the sac (fig. 1). The tip being held well against the bone, the handle of the probe is raised until it is directed straight down; it is kept close to the bony wall until it reaches the bottom of the sac, when it is turned at an angle of 45 degrees and forced through the lateral nasal wall into the nasal cavity (fig. 2). It should enter just in front of the anterior tip of the middle turbinate, which is identified by inspection of the nasal chamber through the nostril.

The probe is now slightly withdrawn in order to make a vertical incision through the mucoperiosteum, which passes through the point of entrance and extends ⅜ inch (1 cm.) above and below (fig. 3). Its edges are elevated and reflected both forward and backward in order to expose the bare bony surface.

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39. Wiener, M., and Sauer, W. E.: New Operation for Relief of Dacryocystitis Through Nasal Route, J. A. M. A. **75**:868 (Sept. 25) 1920.

40. Sauer, W. E.: Dacryorhinocystotomy: Combined Methods, Ann. Otol., Rhin. & Laryng. **32**:25, 1923.

41. Wood, V. V.: Intranasal Surgical Treatment of Chronic Dacryocystitis, Tr. Am. Laryng., Rhin. & Otol. Soc. **39**:554, 1933.

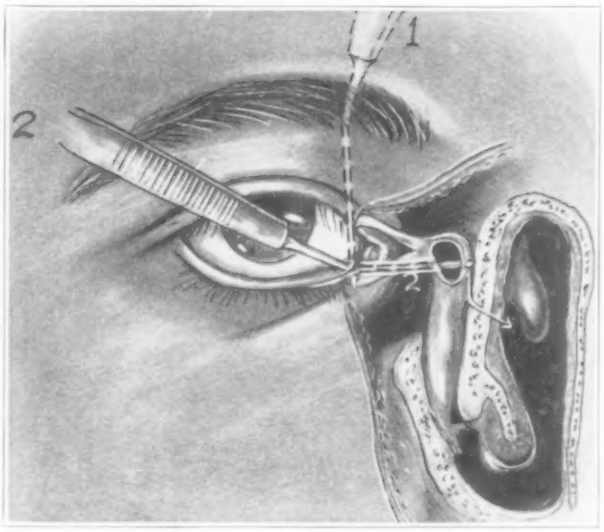


Fig. 1.—Ziegler probe passed into the punctum and along the canaliculus as far as the sac.

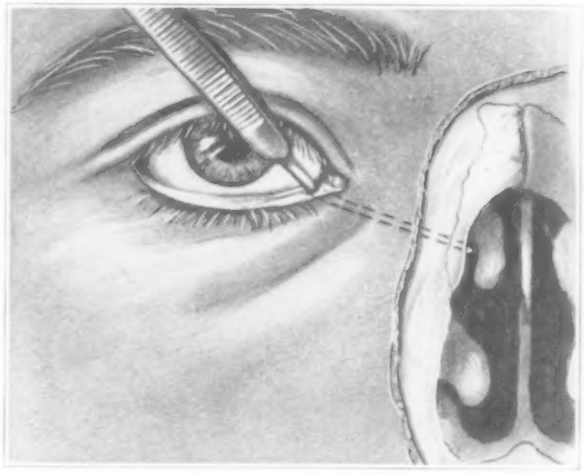


Fig. 2.—Probe forced through the lateral nasal wall into the nasal cavity.

The probe is then pushed into the opening in order to determine exactly its location. An electrically driven burr 2 mm. in diameter (fig. 4) follows its disappearing tip and enlarges the opening in the bone until it attains a diameter of 8 to 10 mm.; it is then extended far enough above to enable the probe to be introduced horizontally into the nose.

A portion of the sac is now pushed through the bony opening into the nasal cavity by means of a Bowman probe (fig. 5), which is now substituted for the Ziegler. With the sac held in this position, biting forceps (fig. 6) are introduced into the nose, and as much of the medial and lower wall of the sac as possible is removed with them.

The point of an Anel syringe is introduced into the canaliculus, and thorough irrigation is carried out through the sac and into the nose. The reflected muco-

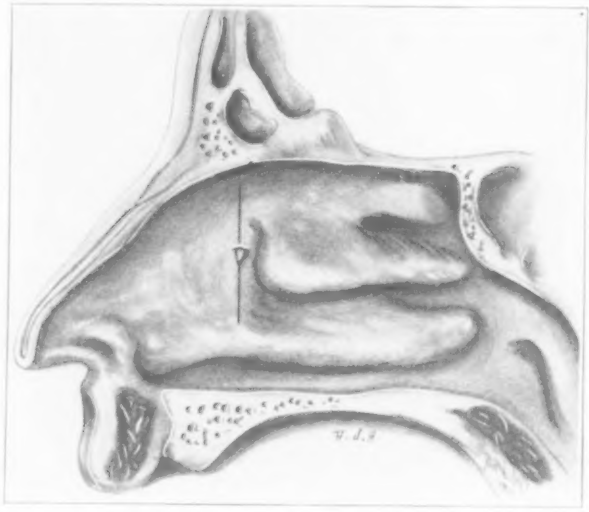


Fig. 3.—Vertical incision through the mucoperiosteum.

periosteum is brought back into place (fig. 7), partially covering the bony opening. This prevents excessive granulations and shortens the time of healing.

Bleeding is usually slight; in no case has packing been required. In some cases a punctum dilator must be used before the Ziegler probe can be introduced. In none of the cases did we find it necessary to slit the punctum. When the middle turbinate is large a portion of the anterior end may have to be removed immediately before the introduction of the probe. If the nose is narrowed by a septal deviation, submucous resection must precede the operation, though in the majority of cases this has not been found necessary.

On the third postoperative day a probe is introduced through the punctum, the canaliculus, the sac and the operative opening into the nose. This procedure may have to be carried out several times afterward. Granulations are kept down by means of a 40 per cent solution of silver nitrate until the wound in the nose is completely healed.

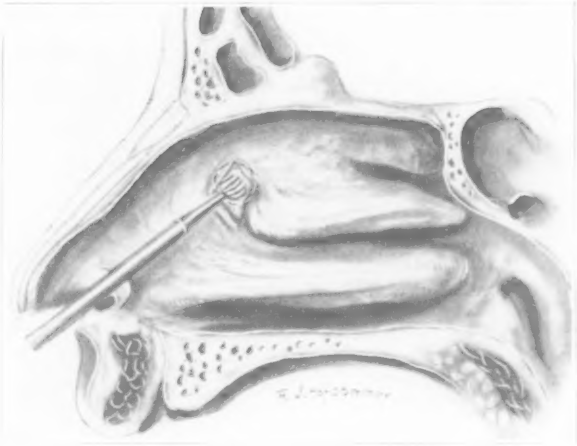


Fig. 4.—Electrically driven burr enlarging the opening in the bone.

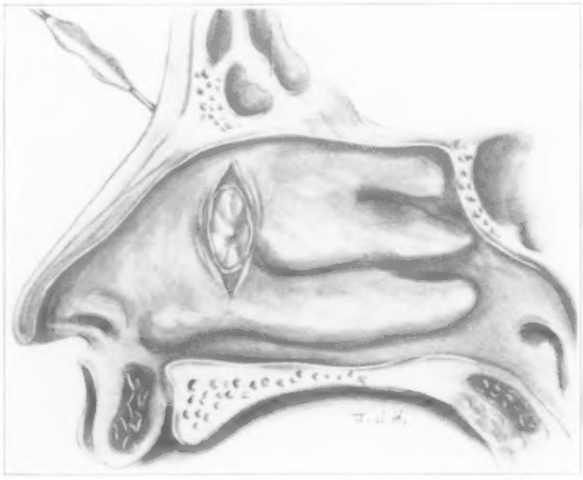


Fig. 5.—Portion of the sac pushed through the bony opening into the nasal cavity by means of a Bowman probe.

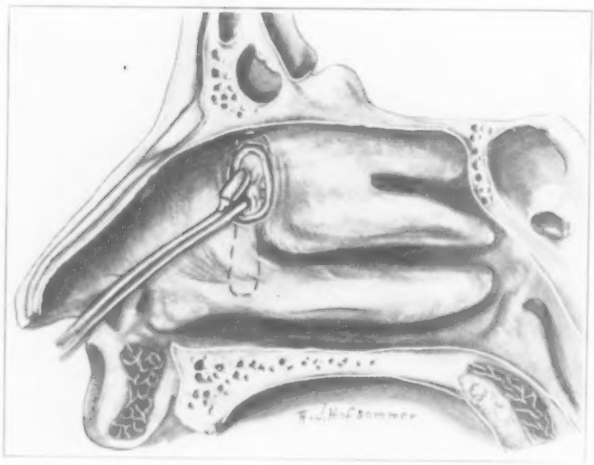


Fig. 6.—Biting forceps removing as much as possible of the medial and lower wall of the sac.

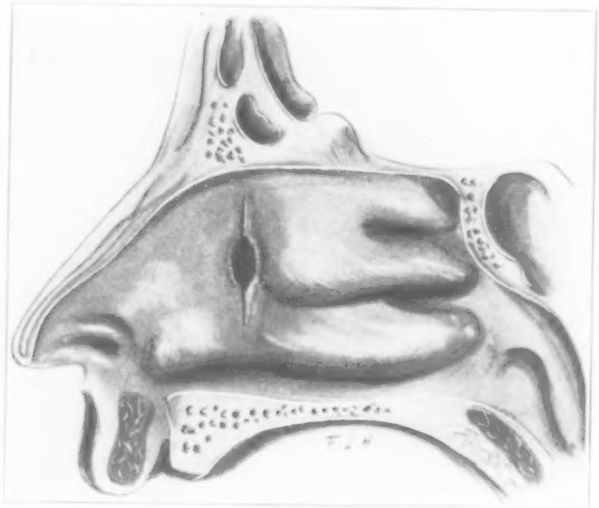


Fig. 7.—Reflected mucoperiosteum brought back into place.

Wiener and I have been following this method since 1911. Some of our patients we have been able to follow for more than twenty years. Our results on the whole have been good. Complete cessation of the purulent discharge and freedom from tearing was secured in nearly every case. In a few, although the purulent discharge subsided, some tearing remained, especially when the patient was exposed to the wind. In a small percentage of cases the result was unsatisfactory, because a permanent opening in the nose could not be maintained. Some of our failures in the beginning were due to our operating, in our enthusiasm to try out the method, in all cases in which local treatment did not produce a response, without due consideration for the pathologic conditions in the lacrimal apparatus, the nose and the paranasal sinuses. We learned that a satisfactory result could not be obtained as long as one or more of the sinuses was chronically infected. It is therefore necessary to study all cases carefully and to eliminate pathologic conditions in the nose and the sinuses before attempting an operative procedure on the lacrimal sac. We now have roentgenograms of the sinuses made, regardless of clinical findings. The roentgen visualization of the nasolacrimal sac according to the method of Hourn [42] has not been satisfactory in our hands. Unfortunately our results have been almost consistently bad in cases in which the bone has been fractured in this area, especially when callus was formed extensively. Failures occurred also when the lacrimal sac happened to be small. For young children with small nasal passages the after-care is a special problem. Nevertheless, we have had a number of cases of young children in which satisfactory results were obtained although the outlook was most discouraging.

The success of the operation depends largely on securing a sufficiently large bony opening into the nose so that the sac can be readily visualized when its nasal wall is pushed into the nose with the Bowman probe. It is also essential that its wall can be grasped with the Hartmann forceps and the greater part of it removed. In other words, one must be able to secure a large enough opening into the sac itself so that there will be no danger of its closing later on.

Our results have been better since we have preserved the mucoperiosteum by reflecting it before enlarging with the burr the opening made by the Ziegler probe. This mucoperiosteum is replaced so that the raw edges of the bony window are covered by the flaps after the operation has been completed. This shortens the time of healing by reducing the area of the granulating surface. Should granulations appear, they are reduced by the application of a 40 per cent solution of silver nitrate, as has always been our practice.

42. Hourn, G. E.: X-Ray Visualization of Naso-Lacrimal Duct, Ann. Otol., Rhin. & Laryng. **46**:962, 1937.

In our opinion this method has all the advantages of other intranasal methods, avoidance of external scar and damage to the capillary action of the canaliculi, plus simplicity, which is lacking in most of them. If necessary the operation can be performed during active inflammation of the sac. In case of failure due to a membranous closure of the opening in the nose the sac can be easily reopened as a simple office procedure. We have done this a second or even a third time in a number of cases, finally securing permanent drainage. When this intranasal operation is not successful it does not interfere with any external operation, such as the Toti or the Mosher operation, or with complete extirpation of the sac.

It is now the opinion of a number of ophthalmologists that the intranasal operation for drainage gives more security as a preliminary step preparatory to intraocular operations such as those for cataract or glaucoma when the sac is inflamed than does extirpation of the sac. Mattice [43] found pneumococci in the conjunctiva in 45 per cent of cases after extirpation. Bumke and West, following Elschnig's method, found that cultures were usually negative within two or three days after the intranasal operation.

DISCUSSION

Dr. Ralph A. Fenton, Portland, Ore.: Mr. Chairman, I should like to ask Dr. Sauer whether he and Dr. Wiener have found it possible to secure good results in this excellent operation, which I may say I have used myself for some twelve years on occasion, in cases in which the lacrimal sac has developed extensions down into the tissues of the face, has become bulging and has certain diverticula, as one might call them. It has been my feeling in following mistakes in technic that probably this class of dacryocystitis cannot be remedied by anything except an external operation, such as the Toti-Mosher, which my associates and I have used for a long time also, and that the excellent procedure of Dr. Sauer and Dr. Weiner must be reserved for cases in which the sac is relatively in its normal position, is not too extensively thickened and can be, as he has shown, pushed into the nose through an excellently made opening in its side wall.

It is true that if the Sauer procedure does not work, one can always do the external operation. But in attempting to operate by the external route after an attempt to do this much simpler procedure, my associates and I have found that the presence of scar tissue due to the earlier procedure made a little trouble in the dissection.

Since this method of operation was discovered, we have abandoned the type of work advocated twenty-five years ago by Dr. West; that, with its extensive instrumentation, is entirely unnecessary, and I feel that Dr. Sauer's procedure has been a great improvement.

Dr. Harris P. Mosher, Boston: I agree heartily with Dr. Sauer that missionary work is still necessary among the ophthalmologists in regard to operations on the lacrimal sac for its chronic infections. Evidently, in Dr. Sauer's hands and Dr. Fenton's hands, this operation has been a success. After hearing

43. Mattice, cited by West, J. M.: The Clinical Results of the Intranasal Tear Sac Operation. Tr. Sect. Ophth., A. M.A., 1931, p. 81.

West's paper and trying that procedure, I gave it up for the external operation. Unintentionally I modified and simplified the Toti operation, thinking I was doing the Toti, and have followed that procedure since. I, however, have always had the feeling that the fundamental principle of surgery was to operate by sight when and where one can. There are cases in which, evidently, Dr. Sauer does operate fully by sight. But I am sure that there are other cases in which he does not and cannot fully operate by sight on account of the anatomic configuration of the nose.

I have had the fashion of saying, to encourage young operators, that the external operation on the lacrimal sac is one of the most satisfactory, from the economic standpoint and from the professional and scientific standpoint, of operations in surgery.

Dr. Charles T. Porter, Boston: My associates and I tried first the intranasal operation with some measure of success in a few cases, but they were not sufficient to warrant carrying on. Frequently, after failures by the ophthalmologist in trying to remove the entire sac or, later on, in trying to do the operation Dr. Mosher developed at the hospital, the patients were turned over to us for the operative procedures to be completed. A split canaliculus and a split punctum leave the sac with a glued-up common punctum. Such patients gave us perhaps more trouble than all the rest, but we finally succeeded in treating them by eliminating the common punctum and leaving sometimes a stilet in the lower one for a while. This complicated condition has given us the most concern.

Dr. W. E. Sauer, St. Louis: In answer to Dr. Fenton, with reference to the pouching of the sac, that suggests one of the advantages of the Ziegler probe, which is a sharp-pointed instrument. One finds the bottom of the sac before one enters the nose.

In reference to Dr. Mosher's remarks about the external operation, my associates and I never attempt an operation on a patient that has been operated on two or three times before by some of the ophthalmologists, but we have seen 1 or 2 patients, possibly more, in the clinic that have been operated on with large keloids; so that is the objection to the external operation. This operation is not a cure-all. One cannot expect success in cases in which there are pouches and in which the sac happens to be small. The important thing is to get rid of the pathologic condition in the nose before one attempts any operation on the lacrimal sac.

Case Reports

PAPILLARY TUMORS OF THE TONSIL

J. Julian Chisolm, M.D., Baltimore

Large benign lymphoid tumors arising from the body of the faucial tonsil are rare. Small pedunculated papillomas or fibromas attached to the tonsils, the faucial pillars or the uvula are common. New [1] in 1931 reported 357 tumors of the tonsils and pharynx, of which 63 were benign. Of the latter, 35 were small pedunculated papillomas, which arose from the pillars in 40 per cent of cases, from the soft palate in 28.6 per cent, from the tonsils in 28.6 per cent and from the posterior wall of the pharynx in 2.8 per cent. There were only 5 true lymphoid tissue tumors, all occurring in women. One of these, a pedunculated growth 3 inches (7.6 cm.) in diameter, attached to the upper pole of the left tonsil and composed of papillary tonsillar tissue, was strikingly similar to that in case 1 of this report. Harold D. Smith [2] stated that in a microscopic study of a series of 2,200 tonsils removed by operation he found 1 papilloma, which showed marked proliferation, spreading out from the capsule. In recent years benign tumors of the tonsils have been reported also by Friend,[3] Kelemen,[4] Pearlman and Pilot,[5] Dunn,[6] Reuys,[7] Zoltan,[8] Oppikofer and Weinhold [9] and Frank.[10] Zoltan described a diffuse papillomatous change in the tonsil which he characterized as tuberous hypertrophy. He stated the belief that such growths may be

1. New, G. B., and Childrey, J. H.: Tumors of the Tonsil and Pharynx (Three Hundred and Fifty-Seven Cases): I. Benign Tumors (Sixty-Three Cases), Arch. Otolaryng. **14**:596 (Nov.) 1931.

2. Smith, H. D.: Microscopic Pathology of the Palatine Tonsil, Arch. Otolaryng. **21**:426 (April) 1935.

3. Friend, L. J.: Fibro-Adenolipoma of the Tonsil, Arch. Otolaryng. **3**:448 (May) 1926.

4. Kelemen, G.: Drei Tonsillentumoren, Ztschr. f. Hals-, Nasen- u. Ohrenh. **16**:556 (Nov. 8) 1926.

5. Pearlman, S. J., and Pilot, I.: Lymphoid Tumors of the Tonsils: Report of Four Cases, Arch. Otolaryng. **5**:143 (Feb.) 1927.

6. Dunn, L. S.: Tumors, Benign and Malignant, of the Tonsil and Peritonsillar Area, Laryngoscope **39**:16 (Jan.) 1929.

7. Reuys, H.: Papillom des Gaumens—Schleimhautplattenepithelcarcinom der Tonsille—Cystadenoma papillare des Nasenvorhofes, Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:167 (Dec. 18) 1932.

8. Zoltan, S.: Ueber die tuberose Hypertrophie der Gaumenmandeln, Monatschr. f. Ohrenh. **69**:864 (July) 1935.

9. Oppikofer, E., and Weinhold, H.: Ueber die papilläre Hyperplasie des lymphatischen Rachenringes, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **144**:135, 1937; "Ueber papilläre Hyperplasie des lymphatischen Rachenringes": Bemerkungen zu den Ausführungen von Dr. Stefan Zoltán, ibid. **144**:347, 1938.

10. Frank, I.: Papilloma of the Tonsil, with Report of Three Cases, Ann. Otol., Rhin. & Laryng. **47**:715 (Sept.) 1938.

induced by stasis, infection or intoxication but expressed feeling that the primary change is hypertrophy followed by stasis, hyperemia and edema. Oppikofer expressed the opinion that papillomatous tumors have an infectious basis.

Frank reported 3 cases and furnished a bibliography of twenty-two articles. He stated that Vidau in 1937 found reports of only 16 cases of tonsillar papillomas in the literature. Frank described the microscopic picture of these benign growths. They are composed of squamous epithelium extending in finger-like, sometimes branching projections about a delicate core of connective tissue without inflammatory changes. Reuys, reporting a papilloma of the soft palate the size of a half dollar in a girl of 4 years, observed that the tumor was characterized histo-

Fig. 1 (case 2).—Low power magnification of a papillary tumor almost completely replacing normal tissue in the upper half of the right tonsil.

logically by extensive and rapid formation of immature connective tissue elements and that mature connective tissue was almost lacking. In addition to papillomas, nonmalignant growths which may be encountered in the tonsils or peritonsillar area include adenomas, fibromas, chondromas, angiomas, dermoid cysts, osteomas, myomas, fibrochondromas, fibrolipomas and fibrolymphadenomas.

REPORT OF CASES

Case 1.—A woman aged 64 was seen on July 25, 1933, complaining of difficulty in breathing at night, first noticed five years previously. At the onset she had consulted her family physician, who told her that the right tonsil was much enlarged. Since then it had gradually increased, and the difficulty in breathing

had progressed to such an extent that she could not sleep except when lying on the right side. The only other symptoms were fulness in the throat, a pulsating sound in the right ear at night and some pain and stiffness in the joints. On examination the only significant findings were in the throat. The left tonsil was of moderate size and showed evidence of chronic infection. There was a large,

Fig. 2 (case 2).—Higher magnification of the area marked in figure 1, showing in cross section, a column of tumor tissue beneath the pharyngeal surface of the tonsil.

rather pedunculated growth arising from the center of the right tonsil and extending beyond the midline. Its surface was nodular and not ulcerated but beneath the epithelium were a number of small cysts containing yellowish material. There were no enlarged glands in the neck. A diagnosis of benign tumor of the right tonsil was made. On August 5 an operation was performed at the Church Home

Fig. 3 (case 2).—High power magnification showing finger-like proliferations of lymphoid tissue covered with stratified squamous epithelium.

and Infirmary with the patient under anesthesia induced by avertin with amylene hydrate and nitrogen monoxide with oxygen. Dr. Grant Ward removed the right tonsil and the tumor in one mass by throwing a tonsil snare around it and applying a strong cutting current to the wire. Remaining tags of tissue in the fossa were coagulated. I then removed the left tonsil by sharp and blunt dissection. There were no postoperative complications and when the patient reported for observation, on December 5, she was free of symptoms and the throat looked clean. A pathologic report by Dr. Vernon L. Norwood was as follows: "This specimen is a mass of tissue 2.5 by 1.5 by 1 cm., which is said to have been removed as a pedunculated growth from the tonsil. The surface presents a smoothly lobulated cryptic appearance similar to that of a normal tonsil. On section numerous cysts filled with yellowish purulent material were found." The diagnosis was papillary hypertrophy of the right tonsil.

Case 2.—A girl aged 4 years was referred by her pediatrician because of nasal obstruction and enlargement of the right tonsil which had been noted for six months. Examination, on April 12, 1938, revealed moderate enlargement of the glands at the angles of the jaws on both sides. The left tonsil was moderately enlarged. The right tonsil was tremendously enlarged and extended well beyond the midline. The surface was lobulated and cryptic and felt soft to palpation. The physical examination otherwise did not reveal any abnormalities. The tonsils and adenoids were removed with the patient under ether anesthesia at the Johns Hopkins Hospital on April 14. The adenoids were small. The left tonsil dissected out easily. The right tonsil proved to be friable, and the tremendous enlargement was confined to the upper pole. It extended deeply beneath the anterior pillar and seemed to be invading the muscle of the tonsillar bed. A definite capsule could not be demonstrated. Bleeding was rather profuse. The operator suspected a sarcoma and removed a portion of the mass for a frozen section. A benign growth was reported. The mass was completely removed then, by sharp and blunt dissection. The only postoperative complication was mild scarlet fever, which developed on the sixth day after operation. The child was examined seven months later. She was free of symptoms, and the throat was clear. The pathologic report was as follows: "The left tonsil is 1.5 cm. in diameter. The right tonsil is 3 cm. in diameter, with unusually large and distinct lobulation. The lymphoid follicles are large, with huge, extremely active germinal centers. A suggestive malignant change is not observed. The innermost layers of epithelium are slightly infiltrated by inflammatory cells. The tonsillar crypts are evidently extremely deep, and several of them are found in cross section in the middle of the section showing small islands of lymphoid tissue surrounded by stratified squamous epithelium, giving the picture almost of a papillomatous tumor" (figs. 1 to 3).

These cases of benign papillary tumor of the tonsil are reported because of the rarity of the condition. Histologically the two growths were similar. One occurred in a woman of 64 and the other in a girl of 4 years. It is conceivable that had a biopsy been made of certain portions of the tumor illustrated, a mistaken diagnosis of malignant tumor might have been made. This point should be kept in mind when clinical observations and the biopsy report are at variance in a case of pharyngeal tumor.

MENINGOCOCCIC CORYZA

L. Dell Henry, M.D., and Hugh A. Kuhn, M.D., Hammond, Ind.

Meningococcic coryza, or rhinitis, is a condition rarely seen. We are unable to find any reports in the literature. That meningococci are carried in the nasopharynx by a fair percentage of normal persons has been established by numerous American and English workers in surveys made both in the army and in civilian life. Bacteriologists and clinicians all report that the cultures must be taken high in the pharynx behind the soft palate. So to find a large number of meningococci in a nasal secretion is unexpected and unusual.

REPORT OF A CASE

A baby girl, 2½ years old, was brought into the office by her mother, who gave a history of bleeding from the nose on several occasions in the past two weeks, accompanied by a profuse discharge and severe sneezing spells. Examination showed the temperature slightly elevated, and the nose was found to be full of mucoid material. There was markedly pale edema of the mucous membrane but only slight inflammation. The vessels of the septum on both sides were dilated. The clinical appearance was that of an allergic nose, plus dilated vessels in Kiesselbach's triangle. Smears were made of the nasal discharge and stained with Wright's stain in preparation for examination for eosinophils. On examination of the smears, however, no eosinophils were found, but some polymorphonuclear leukocytes and many diplococci were seen. Examination of other smears, stained by Gram's method, showed an abundance of gram-negative diplococci, morphologically like the meningococcus. Both intracellular and extracellular organisms were found. Further bacteriologic studies revealed practically a pure culture of these gram-negative diplococci, which were agglutinated by meningococcus antiserum in a dilution of 1:320.

On questioning it was learned that there were 2 other children in the family, both of whom had had "colds," but a history of nosebleed was not given for these two children. On request, the other 2 children were brought into the office for examination. They were 15 months and 4 years old, respectively.

Examination of these 2 children gave essentially the same results: slight elevations in temperature, profuse nasal mucopurulent discharge, slight inflammation of the mucous membrane of the nose and enlargement of the tonsils but not injection of the pharynx. There was less edema of the nasal mucous membrane in these 2 children than in the first child. Smears and cultures revealed meningococcus the same as in the original case.

For all 3 children sulfanilamide was prescribed, and they were kept under close observation and in isolation. At no time did any symptoms simulating meningitis appear. Six days after treatment had been instituted, the children were all clinically well. Repeated cultures were taken after this time from the nose and nasopharynx, and meningococci were not found.

Cultures taken from the mother on two occasions did not yield meningococci.

COMMENT

It seems that children having such an apparently severe infection, if one may judge this by the numbers of organisms seen in the smears made from the discharge and the amount of discharge present, should be clinically more ill than these children were. They were up and around with no loss of appetite or of energy. Clinically these children appeared like allergic patients except for the bleeding from the nose, and that was easily explained on the basis of the large and exposed vessels in Kisselbach's triangle or could have been due to possible diphtheritic infection. If the condition were allergic the management, naturally, would be vastly different from what it would be if it were nasal diphtheria, meningococcic infection or infection with one of the many other infectious or contagious bacterial agents.

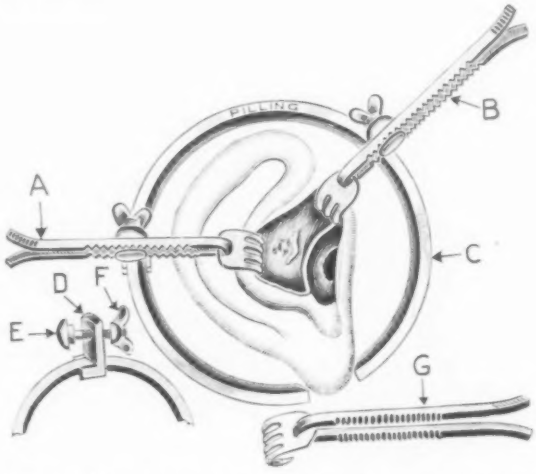
We report this case not only because of its unusualness but to bring out the importance of making smears and cultures from nasal secretions in children for the purpose of differential diagnosis.

Clinical Notes; New Instruments and Technic

MASTOID RETRACTOR, SELF RETAINING, FOR THE ENDAURAL, ANTAURICULAR TECHNIC

BENJAMIN H. SHUSTER, M.D., PHILADELPHIA

A number of otologists have adopted the endaural method of approach to the mastoid described by Dr. Julius Lempert, of New York. A number of them employ this method of approach occasionally. Some of them are doing fistulization for deafness as described by Dr. Lempert, in which case the endaural approach is practically indispensable.



Mastoid retractor, self retaining, for the endaural, antauricular technic. *G*, represents the retractor; *A*, the retractor open at one end with provision for pulling; *B*, the rachet to fix the retractor; *C*, the piston ring around which the retractors are moved to the proper position; *D*, the carrier for the retractor; *E*, the head of the set screw with a projection to fit into the rachet, and *F*, the fly nut to tighten the retractors.

One of the inconveniences of this method is the lack of a self-retaining retractor on the market which would serve satisfactorily. An assistant, therefore, is employed solely for the purpose of holding hand retractors throughout the operation.

The operation for deafness requires many hours, and it is an ordeal for an assistant to sit through it holding the retractors. In this approach it is necessary to shift retractors from one point to another. Therefore, none of the retractors on the market which are fixed at one point serve the purpose. These retractors also

Presented at a meeting of the Section of Otolaryngology at the College of Physicians, Philadelphia, Feb. 15, 1939.

From the Department of Otology, Graduate School of Medicine, University of Pennsylvania, Service of Dr. George M. Coates.

fail to spread the incision in the proper directions. Since I had observed and performed mastoidectomies through this approach, it occurred to me that traction is constantly within the radius of a circle and also that any shift of the retractors places them in another radius of the same circle. A retractor has, therefore, been devised to answer the problem of shifting the retractors within various radiuses of a circle.

A piston ring and retractors with prongs similar to those used by Dr. Lempert but lengthened to suit the purpose were obtained. The stems of the prongs were built like the prongs of a tuning fork which could slide through a set screw and hold in place at various points. Grooves were made on the side of the prongs to keep them from slipping backward and forward. The brackets on which the prongs were placed were tunneled to fit the piston ring. When it is necessary to shift the radius within the circle, a set screw is loosened, the retractor is moved forward, and the brackets may be pushed around the circle to another radius. In this way traction can be made in any radius of the circle without the necessity of an assistant constantly holding and making traction on them, which, as has been stated, may have to be done for hours. An illustration accompanies this description.

The retractors are made by The George P. Pilling and Son Company Philadelphia.

1824 Pine Street.

CATHETERIZING THE SPHENOID

FRED W. DIXON, M.D., CLEVELAND

One is frequently called on to determine the presence or absence of infection in the sphenoid sinus. Because of its location, roentgen reports are often misleading. It therefore becomes imperative to attempt catheterization, either for irrigation or for the instillation of an opaque oil. For this reason a survey of 300 nonmacerated skulls was made to determine the percentage in which the sphenoid sinus could be catheterized, the reason for failure and some data that would make failure less common. It is self evident that if one could locate the ostium and have some way of knowing one has entered the sphenoid cavity irrigation would be easy.

In a recent study of 1,600 macerated skulls,[1] it was found that the bony ostium is either round or oval, the diameter of the round bony ostium being 5.03 mm., while the oval ostium measures 4.02 mm. by 5.8 mm. The membranous ostium conforms in shape to the bony ostium but is much smaller, often almost completely closing the bony opening. There are approximately twice as many round ostiums as oval. The membranous ostium varies from the size of a pin point to 8 mm. in diameter, the average diameter of the membranous round ostium being 3 mm. and the measurements of the oval 2.4 mm. by 4 mm. In most of the cases the mucosa would be traumatized on catheterization, the membranous web occasionally being punctured.

In the macerated skulls, the average distance from the anterior nasal spine to the ostium of the sphenoid sinus in the male was 60.7 mm., and in the female, 57.2 mm. In the living and in the nonmacerated skulls 10 mm. should be added to this measurement for the soft parts encountered at the anterior nares.

1. Dixon, F. W.: A Comparative Study of the Sphenoid Sinus, Ann. Otol., Rhin. & Laryng. **46**:687 (Sept.) 1937.

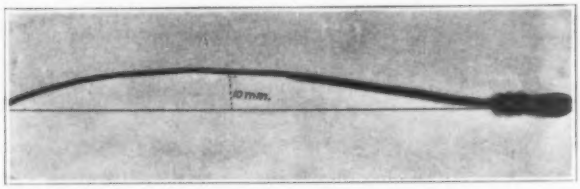


Fig. 1.—A flexible sphenoid catheter bent to conform to the curve of the lateral nasal wall. The shoulder is 7 cm. from the tip.

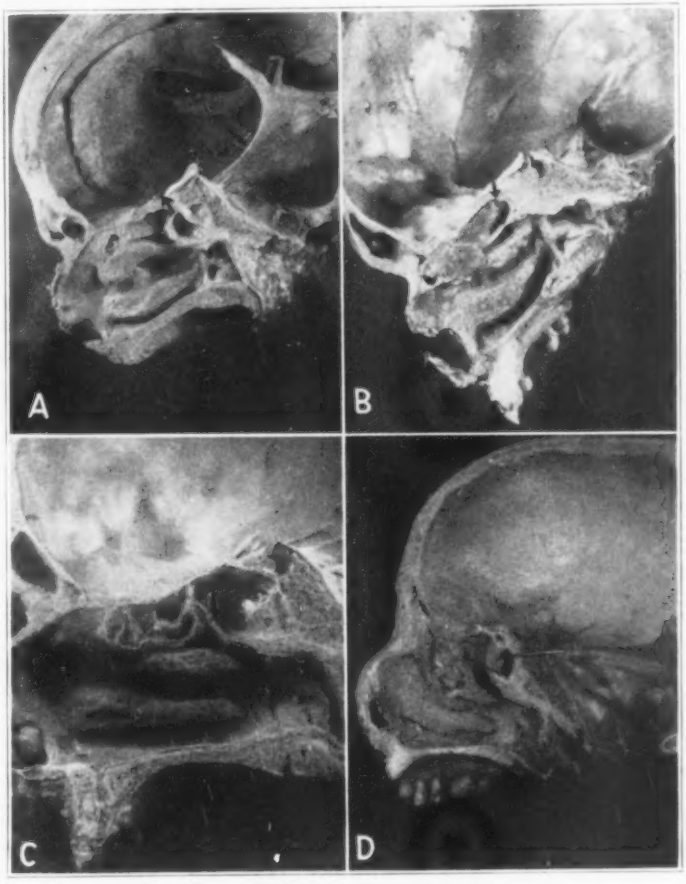


Fig. 2.—*A*, the ostium of the sphenoid sinus pointing *upward*. A catheter introduced in the usual manner would probably slide over the ostium. If force were used, the brain cavity might be punctured. *B*, the ostium pointing *inward*. A small right sphenoid sinus, capacity 0.5 cc., empties by a circular route through the posterior tip of the superior turbinate. *C*, ostium pointing *downward*. The sphenoid cavity is enormous, with thin walls. A probe is in place, pointing downward. *D*, ostium pointing *outward*. Owing to an unusual arrangement of the ethmoid cells, the sphenoid sinus has developed by itself.

The opening of the sphenoid sinus is usually in the upper mesial fourth of the anterior wall of the sinus. The distance from the septum to the center of the ostium varies from 1 to 15 mm., the average distance being 4.92 mm. The distance from the cribriform plate to the center of the membranous ostium also varies from 1 to 15 mm., the average being 8.25 mm.

The most common exception to the location given is when a large postethmoid or ethmosphenoid cell occupies the superior portion of the bone and pushes down

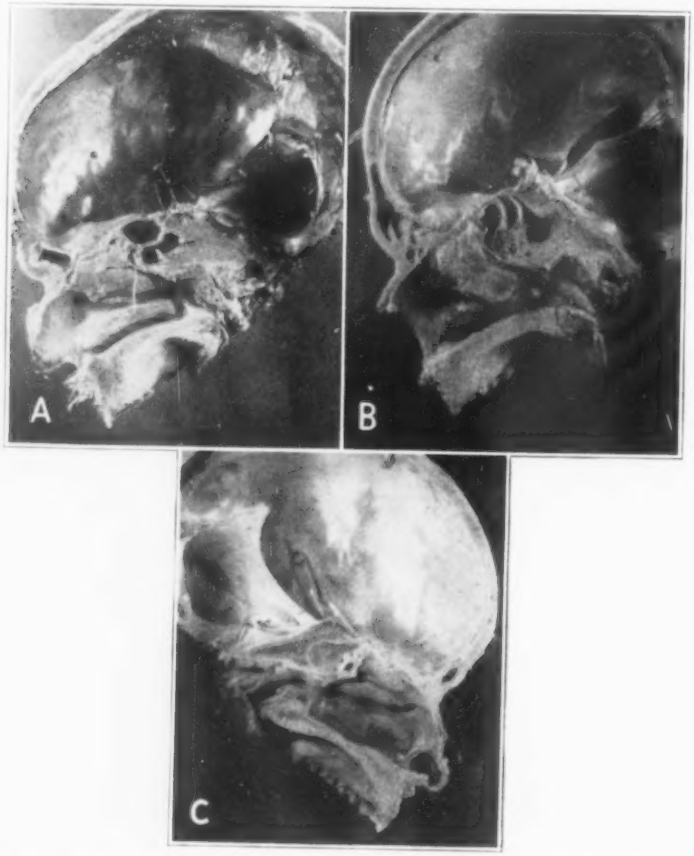


Fig. 3.—*A*, a superior and inferior sphenoid cell, each with its own separate ostium. *B*, an unusual arrangement of posterior ethmoid and sphenoid cells, each having a separate ostium. The sphenoid sinus is impossible to catheterize. *C*, a small *left* sphenoid sinus almost completely surrounded by the *right* sphenoid sinus. The capacity of the left sphenoid sinus is 0.5 cc. The capacity of the right sphenoid sinus is 7 cc. It was possible to catheterize both sphenoid sinuses. The left is so small that one is unable to tell whether or not the catheter is in the cavity.

the sphenoid cell with its ostium. Here the ostium is usually found close to the ethmoseptal angle, approximately 2.4 mm. from the angle.

In an attempt to irrigate the 600 sphenoid sinuses in this group, the sphenoid cavity was entered either by catheterization or puncture in 75 per cent of the cases,

with 25 per cent failures. In 7 per cent of the attempts listed as successful the sphenoid sinuses were so shallow or the ostium so close to the roof that it was uncertain whether the sinus had been entered. Therefore, in only 70 per cent of the cases could one be positive that the efforts were successful. These skulls were all fresh specimens, and, I believe, the percentage of success corresponds closely to that met in actual practice. Dr. Oscar Batson, in a personal communication, expressed the opinion that this percentage of success is too high. If time and care are taken in anesthetizing and shrinking the nasal mucosa, it will be possible to irrigate the sphenoid cavity in the majority of cases. It should always be attempted in patients under suspicion. A roentgenogram should be taken when puncture of the anterior wall of the sphenoid sinus is contemplated. This wall may be dense bone, several millimeters thick, or may be of egg shell consistency, separating the ethmosphenoid recess from the dura.

A long thin flexible catheter with a shoulder 7 cm. from the tip is preferred. This is bent so that the greatest curve is approximately 10 mm. high. In many instances this curve will be too great, but it should conform to the curve of the middle turbinate, bisecting its anterior tip. I believe that this gently curved cannula is better than those bent at an angle similar to the bend of a eustachian catheter, because, first, the turbinates are so shaped, second, it allows one greater leeway in one's search for the ostium and, finally, if one enters the sphenoid sinus one can by slightly rotating the catheter further enter the cavity and thus more thoroughly irrigate.

Various writers in the past have stressed the size of the ethmosphenoid recess as determining the success or failure of catheterization. I believe that a large recess is helpful, but, I feel, success depends more on the angle at which the ethmoid joins the septum. This one can tell by means of the probe. The average ethmoseptal angle is 60 degrees. If the angle is acute, one's chances of success are diminished. A deflected septum interferes with catheterization on the side of the deflection but increases the angle on the opposite side and there facilitates catheterization. The failures were mainly in cases of an ethmoseptal angle of less than 30 degrees. If the anterior wall of the sphenoid sinus is perpendicular or faces down, the ostium probably faces forward. If the anterior wall slopes backward toward the cribriform plate, the ostium faces upward.

Figures 2 and 3 illustrate abnormally placed ostiums.

The reasons for failure in most cases were as follows:

1. A deflected septum interfered with the passage of the catheter.
2. The ethmoseptal angle was too sharp.
3. The ostium was abnormally placed.

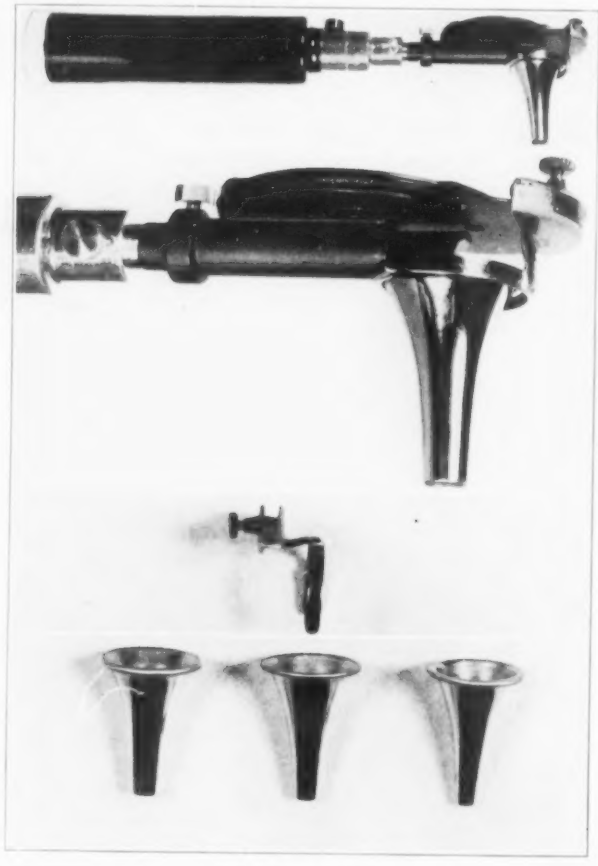
1027 Rose Building.

OTOSCOPIC ATTACHMENT

W. M. Fitzhugh Jr., M.D., and C. E. Hass, M.D., San Francisco
Assistants in Surgery, Leland Stanford Junior University Medical School

The purpose of this paper is to introduce to the medical profession a new instrument which makes an ophthalmoscope into an otoscope. At present the

ordinary diagnostic electric equipment is expensive,[1] and we feel, therefore, that it is essential that the ophthalmoscope should be adapted to as many uses as possible. Heretofore a special otoscopic head has had to be purchased, which has made the carrying case bulky. Our attachment is a compact clip which fits over the top edge of the ophthalmoscope, whose lenses are utilized in the regular way to obtain the necessary magnification and focus of the different parts of the external auditory canal. This device will not interfere with examination of the fundi, it being necessary to remove only the Toynbee ear speculum from the clip.



Photograph of the attachment.

The one disadvantage is that there is no aperture to perform myringotomies, but this deficiency is made up by the following facts: (1) The magnification of any part of the external auditory canal is far greater than that attained with the usual electric otoscope, and (2) the attachment is more compact and less expensive.

Carl Reiner, of Vienna, improved this instrument, making the clip separable.

1. The present prices of the various otoscopic heads range between $12 and $15. Our attachment can be retailed for $2 to $3.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

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CHRONIC PROGRESSIVE DEAFNESS, INCLUDING OTOSCLEROSIS AND DISEASES OF THE INNER EAR

GEORGE E. SHAMBAUGH JR., M.D.
CHICAGO

The most important contribution on otosclerosis for 1938 is the report of the restoration of serviceable hearing in a large proportion of patients operated on by a new surgical procedure. An unusual number of excellent contributions on Ménière's disease appeared in the world literature for 1938.

An attempt is made in this summary to include only articles which bring something new to the knowledge of progressive deafness. My editorial comments are in brackets. The material is arranged, as in last year's summary, in the following order:

Otosclerosis
- Pathology and Etiology
- Treatment

Labyrinthine Deafness
- Pathology of Nerve Deafness
- Hereditary Nerve Deafness
- Traumatic Nerve Deafness
- Nerve Deafness from Drugs
- Nerve Deafness from Infections
- Nerve Deafness from Allergy
- Nerve Deafness from Miscellaneous Causes
- Treatment of Nerve Deafness
- Ménière's Syndrome

OTOSCLEROSIS

Pathology and Etiology.—Nager and Fraser [1] describe the unusual finding of formation of new bone in the scala tympani in 6 otosclerotic patients. They call attention to the fact that as a rule the inner ear

1. Nager, F. R., and Fraser, J. S.: On Bone Formation in the Scala Tympani of Otosclerosis, J. Laryng. & Otol. **53**:173-183 (March) 1938. Nager, F. R.: Ueber Labyrinthveränderungen bei Otosklerose, Schweiz. med. Wchnschr. **1**:85-86, 1938.

shows little or no change in otosclerosis, the pathologic condition being confined to the labyrinthine capsule. In each of the 6 cases in which new bone was found in the scala tympani the otosclerotic process was extensive, involving both windows and often with foci in other parts of the labyrinthine capsule, such as the regions around the semicircular canals, in the internal meatus and around the cochlear aqueduct. In each case the otosclerotic focus had extended to the endosteum of the scala tympani and the newly formed bone was attached to the otosclerotic focus. This new bone was lamellar, and in 4 of the 6 cases the otosclerotic bone had begun to invade the newly formed lamellar bone. The authors conclude that the formation of new bone in the scala tympani is the result of irritation of the endosteum by the otosclerotic process.

Bast [2] continues his fundamental studies of the development of the otic capsule with a description of the fossula post fenestram. This structure, less familiar to most otologists than the fissula ante fenestram, consists of a small evagination of connective tissue from the vestibule into the bony capsule of the labyrinth just posterior to the oval window. It is comparable to the fissula ante fenestram in character and content. It differs from the fissula in that its shape is more saclike and, as a rule, it does not extend through to the middle ear, although in a few cases it does communicate with both the vestibule and the middle ear. The fossula is also less constant than the fissula, being found in only two thirds of the ears in the fetus and early childhood. Tracing the development of the fossula from its earliest evidence in the 8 week embryo into early childhood, the author concludes that it is a more rudimentary and less constant structure than the fissula but that, like the fissula, it represents an area of instability where, during the process of obliteration, calcification and formation of cartilage and new bone occur, the new bone resembling otosclerotic bone. The author concludes that the fissula frequently and the fossula less frequently are the site of the formation of new bone that may represent the first stage in the formation of otosclerotic bone. This accounts for the occurrence of otosclerotic bone usually anterior to the stapes.

[Comment: Bast has given the best explanation for the origin of otosclerotic foci yet offered. He has not explained why the focus that begins in the region of the fissula ante fenestram or, less often, around the fossula post fenestram should in some cases grow and invade the surrounding labyrinthine capsule, leading eventually in a small proportion of cases to ankylosis of the stapes and deafness.]

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2. Bast, T. H.: Development of the Otic Capsule: The Fossula Post Fenestram, Arch. Otolaryng. **27**:402-412 (April) 1938.

Anson, Karabin and Martin [3] trace the development of the stapes and the region of the oval window from their earliest appearance to their adult form. The stapes is first discernible as a fairly distinct ring of cartilage in the human embryo of 7½ weeks. At 10 weeks it is becoming stirrup shaped, and at 19½ weeks it has approximated adult size and ossification begins. The oval window also reaches its adult size and shape at this early age, and the fissula ante fenestram and the fossula post fenestram, when present, are surrounded by cartilage, which at this age is directly continuous with the margins of the oval window. Subsequently the intervening space becomes ossified, but both the fissula and the oval window retain their embryonic cartilaginous rim throughout life.

Nager [4] briefly reviews the pathology of bone of the labyrinthine capsule. Of interest is the statement that in fractures the periosteal layer of the capsule heals normally, while the endochondral layer never heals by bony union, since it cannot form new bone. The histologic picture of ostitis fibrosa of Recklinghausen and of Paget's disease in the temporal bone is closely related to otosclerosis.

Meller [5] describes the temporal bone of a patient with renal rickets. In this disease of children chronic nephritis is combined with osseous changes of rickets. The mastoid process, petrous apex and periosteal capsule showed changes resembling rickets, consisting of the formation of wide osteoid spaces, whereas the endochondral and endosteal layers of the capsule remained almost free from changes.

[Comment: The relative resistance to systemic influences of the part of the capsule most involved in otosclerosis speaks against a general metabolic cause for otosclerosis and in favor of a purely local cause, such as has been indicated by Bast.]

Wilson [6] presents an interesting discussion of the various theories of the cause of otosclerosis. There are about six important theories, the best known being that of Wittmaack, who stated the belief that otosclerosis is due to venous stasis in the labyrinthine capsule resulting in osteoporosis. He based his theory on experimentally produced stasis in chickens. However, not all histologists agree that the changes observed in chickens are identical with otosclerosis; secondly, Wittmaack was not

3. Anson, B. J.; Karabin, J. E., and Martin, J.: Stapes, Fissula Ante Fenestram and Associated Structures in Man: From the Embryo of Seven Weeks to That of Twenty-One Weeks, Arch. Otolaryng. **28**:676-697 (Nov.) 1938.

4. Nager, F. R.: Ueber die Knochenpathologie der Labyrinthkapsel, Acta oto-laryng. **26**:127-137, 1938.

5. Meller, H.: Die Veränderungen des Schläfenbeins bei Rachitis und "renaler Rachitis," Monatschr. f. Ohrenh. **72**:639-671 (July) 1938.

6. Wilson, J. G.: Present Status of the Problem of Otosclerosis, Arch. Otolaryng. **28**:946-953 (Dec.) 1938.

able to produce these changes in monkeys, and, finally, he failed to explain the venous stasis that according to his assumption is present in otosclerotic patients.

The second theory is that of O. Mayer, who described otosclerosis as a reaction to mechanical strain. He stated the belief that the fissula ante fenestram is a synchondrosis or joint to compensate for tensions between the cochlea and the semicircular canals and that the otosclerotic focus begins as porous bone to meet the mechanical strain. Against this theory Bast pointed out that the structure of the fissula is not comparable to synchondrosis. Also it does not explain why the fissula ante fenestram is found only in man.

Gray suggested that otosclerosis is the result of insufficient blood supply to all the nerves in the organ of hearing, basing his theory on the observation that the degree of deafness bears little relation to the extent of the osseous disease and that the severity of the tinnitus is not related to the duration.

Fraser stressed the influence of catarrhal and suppurative otitis media as a factor inciting to otosclerosis in persons with a hereditary tendency. This fails to explain the occurrence in many cases of otosclerosis with no evidence of inflammation of the middle ear, past or present.

Nager and M. Meyer, impressed by the histologic similarity between otosclerosis and Paget's disease, described otosclerosis as localized osteodystrophia.

The author (Wilson) points out that the otic capsule differs from normal bone elsewhere in the body in that it is essentially immature bone. The capsule, like all bone, develops originally from connective tissue, which changes first into cartilage and later into bone. This primary endochondral bone is replaced everywhere else in the body by lamellar bone around haversian canals, but in the endochondral layer of the capsule it persists and is characterized by its weblike strands and the cartilage islands that remain embedded in it. The author agrees with Bast that the fissula ante fenestram represents an area of unstable bone. He suggests that, since the main blood supply of this area is from the middle ear, abnormal developments may occur in it as a result of disease of the middle ear. Thus otosclerosis may be regarded as an inherited susceptibility to environmental influences.

Ruttin [7] reports the unusual finding of a bilateral fistula symptom in a case of otosclerosis. There seemed no doubt concerning the accuracy of the diagnosis of otosclerosis, the clinical picture being typical. The author suggests that the otosclerotic process had destroyed the bone

7. Ruttin, E.: Ueber Fistelsymptom bei Otosklerose, Pract. oto-rhino-laryng. **1**:410-415, 1938.

around the oval window replacing it with connective tissue, thus rendering the stapes abnormally mobile.

[Comment: How does the author explain the deafness if the stapes was still mobile?]

Sommerfeld,[8] impressed by the apparent relation between otosclerosis and the "pregnancy hormones" (suggested by such factors as the predominance in females and the influence of pregnancy and the menopause), injected the urine from pregnant women into 3 guinea pigs. Comparing the labyrinthine capsules of these guinea pigs with those of controls he observed incomplete ossification with large cartilage rests and marked vascularity of the endochondral layer in the guinea pigs which received the injection. He wisely concludes that further confirmation and study are needed before any conclusions can be drawn.

Treatment.—A valuable and significant study of the results of treatment of otosclerosis is contributed by Suggit.[9] Having previously obtained some improvement in hearing in a proportion of cases of otosclerosis by the intratympanic injection of thyroxin or physiologic solution of sodium chloride, he checked his results by injecting the latter through the eustachian tube in 11 cases of otosclerosis while 15 cases of otosclerosis were simultaneously observed audiometrically without any treatment. Improvements in hearing occurred with equal frequency in both groups of cases and averaged up to 10 decibels throughout the tonal range. Since no greater improvement was obtained by the intratympanic injection of thyroxin than by the use of physiologic solution of sodium chloride or by no treatment at all, the author concludes that intratympanic medication for otosclerosis has no real value and that any apparent results are within the limits of normal variation. He contends that "No treatment of otosclerosis can be considered of value unless it can produce a sustained improvement . . . greater than ten decibels over the greater part of the range, 64 to 8,192 d. v. s."

Goldstein [10] reports his results with intratympanic injection of thyroxin for a series of 42 otosclerotic patients during the past three years. The technic described by Gray was followed in every detail. Of the 42 patients, 9 showed no improvement; 10 showed comparatively slight changes with recession six months later, and 23 showed in the audiogram definite improvement maintained six months after treatment.

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8. Sommerfeld, W. S.: À propos de l'otospongiose et de l'influence des hormones de la grossesse sur la capsule osseuse labyrinthique des cabayes, Rev. de laryng. **58**:389-391 (April) 1938.

9. Suggit, S.: Observations on the Variation in Hearing in Otosclerosis, J. Laryng. & Otol. **53**:294-312 (May) 1938.

10. Goldstein, M. A.: Thyroxine Therapy in Otosclerosis: Report of Forty-Two Cases, J. Laryng. & Otol. **53**:444-457 (July) 1938; Laryngoscope **48**:443-457 (July) 1938.

[Comment: These results are at variance with the negative results reported by Livingstone, who continued Gray's series of cases after the latter's death, and by Suggit, who used controls in the form of patients treated with physiologic solution of sodium chloride and others receiving no treatment (see the articles reported earlier in this review and in the review for 1937). On examining the author's results more closely, one finds that audiogram readings before and after treatment are given for 8 of the 23 patients that were improved. Presumably these were the best 8. Analysis of the hearing for the frequencies of 512, 1024 and 2048 discloses an average change of less than 10 decibels for 4 of the 8 patients, and this change was a loss as often as it was a gain. Of the remaining 4 patients, only 1 experienced an improvement of more than 10 decibels in both ears (in each case both ears were treated), and this patient had definite hypothyroidism. It seems likely that the results reported by the author lie within the limits of normal variation with but few exceptions and that several of these exceptions were in definitely hypothyroid patients, so that the possibility that absorption of thyroxin into the system caused the improvement in hearing cannot be excluded entirely.]

A few more reports on the medical treatment of otosclerosis may be briefly mentioned. Kobrak [11] believes that in a certain proportion of cases otosclerosis is due to spasmophilia of the muscles of the middle ear. At first there is rigidity of the stapes and later an ankylosis secondary to the spasm of the muscles. [On the basis of his rather fanciful theory] the author suggests the use of calcium, phosphorus, vitamin D and parathyroid extract in these cases of otosclerosis.

Maggiorotti,[12] regarding ankylosis of the stapes as a joint disease, has used daily intramuscular injections of a glycerophosphate solution which is used for arthropathies. The results as far as the tinnitus is concerned are stated to be encouraging.

Desjardins [13] revives the use of roentgen rays for otosclerosis. Ostensibly in many cases otosclerosis was favorably influenced.

Seiferth [14] (in one instance with the coauthorship of Kolb) contributes two more articles on the treatment of otosclerosis with a deriva-

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11. Kobrak, F.: Pathologische Physiologie als Bundlage praktischer Ohrenheilkunde: I. Zur pathologischen Physiologie der Otosklerose. (Die spasmophile Form der Otosklerose), Pract. oto-rhino-laryng. **1**:186-202, 1938.

12. Maggiorotti, U.: Terapia dell'otospongiosi con le alte dosi di glicerofosfati. Nota preventiva, Valsalva **14**:48-49 (Jan.) 1938.

13. Desjardins, A. U.: Action des rayons X et du radium sur l'oeil et l'oreille, J. de radiol. et d'électrol. **22**:305-317 (July) 1938.

14. Seiferth, L. B.: Ueber die Ätiologie und Behandlung der Otosklerose, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **144**:367-383, 1938. Seiferth, L. B., and Kolb, H.: Ueber die Ätiologie und Pathogenese der Otosklerose auf Grund tierexperimentellen Versuche mit A. T. 10. Ein Beitrag zur experimentellen Otosklerose, ibid. **145**:391-419, 1938.

tive of ergosterol, dihydrotachysterol. He believes that he has shown that the calcium content of the blood is slightly increased in otosclerosis. Furthermore, on feeding this drug to dogs the author observed foci in the temporal bone, chiefly in the external bony canal and outer wall of the middle ear, resembling otosclerosis.

Escat [15] has employed phosphorus therapy for otosclerosis for many years and since 1913 has added pituitary extract on the theory that the hypophysis is the controlling gland of the endocrine system. Of 223 patients so treated, 98 were unchanged, 78 slightly improved and 28 markedly improved, while 19 became worse in spite of treatment. On the theory that the ovaries influence otosclerosis, he also irradiates the ovaries in certain cases with good results. [Since his results, like those of the preceding four authors, were not controlled audiometrically, they must be taken with reservations and regarded as lying within the normal limits of variability of the disease.]

Hughson [16] reports his results in 18 cases of deafness treated by blocking the round window niche with a tissue graft. All but 2 of these have been observed for three or more months after operation, the longest period of observation being twenty months. In no case has the hearing been further impaired by operation except for a brief period immediately postoperatively. In 2 of the 18 improvement was not shown, while in the remainder improvement occurred sometimes immediately and sometimes one to seven months after operation. The average improvement in all 18 cases was 10 decibels for the critical frequencies, a maximum improvement of 20 decibels being obtained in 1 case. Nerve and conduction deafness are equally suitable for this operation. In the discussion that follows this paper, D. E. S. Wishart points out that the author measures improvement by taking the best reading for each tone from a large series of audiograms. When the audiogram taken before operation is compared with the last audiogram, in no case was there either an improvement or an impairment greater than 10 decibels, and this lies within the normal variation. In comparing the ear operated on and the other ear, the same degree of change was observed in both ears in 7 of 8 cases, indicating that the patients' hearing had not been altered by the operation.

Sourdille [17] describes his present technic in the surgical treatment of otosclerosis, which is essentially the same as in previous articles,

15. Escat, A.: Thérapeutique anti-dysovarique de l'otospongiose. Opotherapie hypophysaire. Radiothérapie ovarienne, Ann. d'oto-laryng., October 1938, pp. 921-928.

16. Hughson, W.: Symposium: What Can Be Done for Chronic Progressive Deafness? Rationale, Technique, Case Reports and Observations with Grafts in the Round Window, Laryngoscope **48**:533-551 (Aug.) 1938.

17. Sourdille, M.: The Present Position of the Surgical Treatment of Otosclerosis, J. Laryng. & Otol. **53**:78-83 (Jan.) 1938.

mentioned in this review for 1937, namely, a three stage procedure. The first stage consists of the removal of the skin from the outer two thirds of the posterosuperior bony canal, allowing the surface to become covered with an epithelized scar. In the second stage mastoidectomy is carried out with removal of the head of the malleus and of the superior bony wall of the canal. The cicatricial tissue from the first operation is used to cover the incus and close the aditus to the horizontal semicircular canal. In the third stage the cicatricial flap is elevated from the semicircular canal, which is now opened with great care so as not to injure the delicate membranous labyrinth. The flap is then replaced over the fistula and acts as an extension of the tympanic membrane. The improvement in hearing is due, the author believes, to the transmission of vibrations from the drum membrane to the labyrinthine fistula, analogous to the normal physiologic process. If the fistula ossifies, a secondary operation is necessary to remove the new bone and restore the hearing. The hearing is, as a rule, definitely improved by this operation, up to 10 or 20 times better than before. A contraindication is a dense mastoid or pneumatization of the zygoma, since opening the zygomatic cells is difficult and is often followed by infection and necrosis of the incus.

[Comment: The best criticism of Sourdille's reports is given by Holmgren in the article reviewed immediately after that of Ledoux.]

Ledoux [18] calls attention to the great technical difficulties of Sourdille's operation for otosclerosis. The resection of the head of the malleus in the second stage requires extraordinary skill. The author suggests that by waiting until the third stage, when the malleus is firmly fixed by the skin flap, the head may be resected without the fear of luxation. The author states that simple opening of the labyrinth without disturbing the ossicles will arrest the progress of otosclerosis in early stages with slight deafness.

[Comment: There is as yet no proof that opening the labyrinth will arrest the progress of the deafness in otosclerosis.]

Holmgren [19] presents the most complete discussion to date of his own work on the surgical treatment of otosclerosis. Between 1920 and 1935 he operated on 35 patients with otosclerosis by the following technic: After a preliminary radical mastoid operation with exposure of the horizontal semicircular canal, a fistula was made into the labyrinth, in the early cases in the promontory, in the later cases into the horizontal canal, and covered with mucoperiosteum. The primary result on hearing was good but in every case there was no permanent result, since the fistula became covered with bone. In 1935 a Thiersch graft was used to cover the fistula and in 1 case gave a good permanent result,

18. Ledoux: Remarques techniques à propos de l'opération de Sourdille, Bull. Soc. belge d'otol., rhin., laryng., 1937, pp. 310-313.

19. Holmgren, G.: The Surgical Treatment of Otosclerosis, Hygiea **100**: 681-723 (Oct. 8); 757-770 (Oct. 15) 1938.

but the wide opening to the outside was troublesome. Therefore, he next covered the fistula with a small strip of fat with a piece of gold leaf intervening to prevent adhesion to the membranous labyrinth. However, although the immediate improvement in hearing was marked, it was not lasting, and reoperation showed new bone covering the fistula. Lately the author has done the operation in two stages: In the first the horizontal canal is trimmed down almost to the lumen and the wound filled with paraffin and sutured. In the second stage the layer of connective tissue that forms over the horizontal canal is used as a flap to cover the fistula. In several cases the fistula remained open; in others it has closed. Because fistulas due to cholesteatoma do not close, the author has covered those in his last 2 cases with the squamous epithelium of a previously created radical cavity. It is too recent to judge the final result.

Since the fistulas close so readily the author states that several fistulas should be made in each case, so that he usually makes a fistula into two or more canals. To avoid injury to the membranous labyrinth the fistula is made as close to the concavity as possible, where the membranous labyrinth lies away from the bony wall. Decompression of the endolymph exposing the saccus endolymphaticus also is done, on theoretic grounds.

At the moment a fistula is made the hearing is considerably improved. In the first days after operation the hearing decreases, and then, when the result is favorable, it increases again. As regards the permanent results, the author states that, since ossification of the fistula usually occurs and no method of preventing it has been found, the good initial result is only temporary in a great number of cases and that occasionally the hearing is worse than before operation.

The author emphasizes that audiometric testing must be used to follow the hearing. Sourdille's published results are severely criticized because of the absence of audiometric determinations and because of the way in which improvement of hearing was measured. Thus, a change from perception of the conversational voice close to the ear to perception at 5 to 10 cm. from the ear is called by Sourdille 5 to 10 times better hearing, while if it is perceived at 20 cm. he considers the patient to hear 20 times better. While one must assume from the degree of improvement in hearing that the fistula has remained open in several of Sourdille's cases, the claim of 74 per cent favorable results is not justified by the published records.

Nager [20] described the histologic observations on a patient who had had an operation for otosclerosis by Holmgren [21] four weeks before

20. Nager: Demonstration der Schmitte einer Patienten mit Otosklerose, Acta oto-laryng. **26**:342 (July) 1938.

21. Holmgren, G.: Ein operierter und an intercurrenter Krankheit gestorbener Fall von Otosklerose, Vorläufige Mitteilung, Acta oto-laryng. **26**:340-342 (July) 1938.

death. A woman of 45 with otosclerosis showed reduction of hearing for the conversational voice to 0.2 meter from the right ear and 0.3 meter from the left ear. The left ear was operated on, fistulas being made into the horizontal and posterior semicircular canals and gold leaf and then fat being used to cover the fistulas. The hearing immediately improved to more than 10 meters and the next day to 35 meters. It then gradually decreased to 6 meters on the fourth postoperative day, 1 meter on the sixth day and 0.5 meter on the twelfth postoperative day. The patient died of a pulmonary embolus twenty-seven days after operation.

Histologic examination showed active otosclerosis with a rich vascular supply and osteoclasts. The operatively created fistulas were partly open but were partly bridged over by osteoid substance, which projected over from the periosteal capsule into the operative cavity. The membranous canal was completely preserved, and the endolymph was only slightly more deeply stained in the canals than elsewhere in the labyrinth. The operative cavity in the mastoid was free from inflammation. The author emphasizes the absence of signs of inflammation in the labyrinth four weeks after fistulization.

[Comment: Neither Holmgren's nor Sourdille's articles present anything not in their previous reports. Holmgren admits that a permanent improvement in hearing after his operation is the exception, and he ascribes his failures to regeneration of bone, which closes the fistulas. Sourdille claims good results from his three or four stage operation, requiring several months between each stage, partly for the infection to subside. However, Sourdille's results have always been obscured by his method of reporting improvement, by his refusal to allow any one to see him operate or to examine any but an exceptional patient and by his failure to record the hearing audiometrically before and after operation. One must conclude, with Holmgren, that Sourdille probably has an occasional permanent fistula with permanent improvement in hearing but that with the available evidence one cannot accept his claim of 74 per cent positive results.]

Lempert [22] reports his results for 23 patients with otosclerosis operated on by a new surgical procedure. The operation is described in the minutest detail with illustrations depicting each step. The endaural approach to the temporal bone previously described by the author is employed with the area under local anesthesia. The operation consists essentially in creating a fistula into the horizontal semicircular canal and covering the fistula with a plastic flap consisting of the skin of the superior and the posterior bony wall of the canal intact with Shrapnell's

22. Lempert, J.: Improvement of Hearing in Cases of Otosclerosis: A New, One Stage Surgical Technic, Arch. Otolaryng. **28**:42-97 (July) 1938.

membrane and the pars tensa. A series of 23 patients operated on by this method is reported in detail with audiograms before and after operation. Four patients had poor bone conduction before operation (indicating loss of nerve function) and the hearing was not improved. In 1 of the 4 the fistula began to show signs of closure at the end of ten days and was completely closed in three weeks (as evidenced by disappearance of the reaction to the fistula test). In 22 patients the fistula remained open and the reaction to the fistula test remained positive to the lightest pressure of a cotton-tipped applicator. The first patient reported on was operated on seven months before the report was made, while the most recent operation was one month before. On the basis of previous experience, before the operation was perfected, the author has found that when regeneration of bone takes place to close a fistula it does so immediately after operation so that by four weeks after operation the reaction to the fistula test becomes negative. When regeneration of bone has not occurred two months after operation, the author believes that the fistula will remain permanently open.

[Comment: Examining the published audiograms, the degree of improvement in hearing in the 19 patients with positive results leaves little doubt concerning the value of this operative procedure in the author's hands. Contrasted to an improvement of less than 10 decibels which characterizes, with rare exceptions, previous reports of the results of treatments of otosclerosis, one sees an improvement for the critical frequencies of 512, 1024, and 2048 that averages 21.4 decibels for the 19 patients reported as experiencing good results. In a number of the cases, however, the opposite ear also showed some improvement, and in 1 case it showed more improvement than the ear operated on (case 15).

Those who have observed the author's patients and who have seen him perform the operation are impressed by the degree of improvement in hearing experienced by the patients, which leaves no doubt concerning the validity of the author's results. The operative procedure itself gives an end result that anatomically is similar to that aimed at by Sourdille, with the important differences that the Lempert operation is a one stage procedure and that the end results obtained by Lempert are positive in a far larger proportion of cases than are those obtained by Sourdille, though comparisons are difficult because of Sourdille's obscure reports. Finally, the complete freedom from postoperative infection in Lempert's cases is in marked contrast to the infection that occurs as a rule in Sourdille's cases, judging by his description. The difference is evidently one of technic, since Lempert employs the most meticulous surgical asepsis, both in his operation and in the postoperative care.

The technical proficiency displayed by Lempert in his operating is an important factor in his results. The creation of the plastic flap without

tearing the delicate attachment of the skin to Shrapnell's membrane is a difficult technical accomplishment. The creation of the fistula without injuring the membranous labyrinth also is a delicate procedure, requiring unusual skill. In the final analysis, the results obtained by Lempert, which have never before been approached, are due to his success in maintaining the patency of the fistula. Further investigation is necessary to show whether it is the use of the polishing burr in making the fistula, the use of Shrapnell's membrane to cover the fistula or both that is responsible for its permanence.]

LABYRINTHINE DEAFNESS

Pathology of Nerve Deafness.—From the otologic laboratory at Johns Hopkins University comes an important study of the histologic findings in a series of cases of perception deafness with loss for the low tones as well as for the high tones. A total of 35 ears was examined histologically by Oda.[23] In each case there was slight to moderate impairment of the hearing for low tones, with marked impairment in most cases for the tones above 1024 vibrations. In each case the 512 vibration tuning fork was heard longer by air than by bone conduction, indicating inner ear deafness. As controls, the author used patients with normal hearing and patients with impairment for high tones only, making observations on the middle ear and the cochlea. In 4 of the 35 ears no lesion was found greater than those in the controls. The author explains this on the basis of limitations of histologic technic or the possibility of central deafness. Of the 35 ears, 14 showed an abrupt drop in perception for the high tones, and 13 of the 14 were found to have atrophy of the organ of Corti in the basal turn. There were 15 ears with a gradual loss of hearing for the high tones, and in all 15 atrophy of the spiral ganglion cells and nerve was noted. These observations are in harmony with those reported in the previous studies of Crowe, Guild and Polvogt, who found these two types of histologic change in these two varieties of nerve deafness for high tones. In 6 ears the audiogram taken before death was of the horizontal type, and in all 6 the prominent lesion was in the middle ear. In none of the 35 ears were there signs of past or present inflammation in the cochlea, the changes being purely atrophic. The author concludes that impairment of hearing for low tones with a positive reaction to the Rinne test is in most cases due to the same changes as impaired hearing for high tones, namely, atrophy of the organ of Corti in the basal turn. It was not possible to demonstrate atrophic changes in the apical turns which could be held responsible for the loss of hearing for low tones. In the 6 ears with the horizontal

23. Oda, D.: Observations on the Pathology of Impaired Hearing for Low Tones, Laryngoscope **48**:765-792 (Nov.) 1938.

type of audiogram a lesion of the middle ear was combined with that of the inner ear. The author believes that while the tone of 8192 is heard in the middle of the lower basal turn, 4096 in the upper end of the lower basal turn and 2048 in the lower part of the upper basal turn, the area for the hearing of lower frequencies is not restricted.

[Comment: The author's findings are in harmony with clinical experience in that nerve deafness is usually permanent. However, there is another type of nerve deafness for low tones, with which the hearing may show remarkable improvement, namely, that in Ménière's syndrome (to be discussed later). This condition must be due to some other type of pathologic change than that described by the author, since an atrophic nerve or organ of Corti could not be expected to regenerate. The pathologic nature of nerve deafness for low tones that is subject to fluctuations and is accompanied by vertigo and diplacusis remains to be discovered.]

Crowe and Guild [24] report the case of a child with impairment of hearing due to hypertrophy of lymphoid tissue around the eustachian orifice; radiation of the nasopharynx resulted in restoration of hearing to normal. Because the audiogram taken before treatment revealed a loss for high tones greater than that for low tones while after treatment the hearing was normal for all tones, the authors conclude that a purely conductive lesion (occlusion of the eustachian tube) may cause a greater loss for high than for low tones, contrary to the usual teaching.

[Comment: I pointed out several years ago, and again last year, that occlusion of the eustachian tube resulting in fluid in the middle ear caused a loss for high tones as well as for low tones, due presumably to blocking of the round window, through which high tones are probably heard, in addition to the blocking of the oval window, through which low tones are conducted. When the oval window alone is occluded, as in otosclerosis, the hearing for high tones remains relatively close to normal, since high tones continue to enter the round window normally, whereas the perception of low tones is markedly impaired. The case reported confirms my previous observation.]

Crowe [25] calls attention to the two practical applications of histologic study in cases of nerve deafness. When the loss of hearing begins at 256 or 512 vibrations and increases gradually toward the higher tones, the lesion is atrophy of the cochlear nerve supplying the basal turn of the cochlea, and the organ of Corti is not affected. When the audiogram shows an abrupt or sharply localized impairment of perception for high

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24. Crowe, S. J., and Guild, S. R.: Impaired Hearing for High Tones, Acta oto-laryng. **26**:138-144 (March) 1938.

25. Crowe, S. J.: Diagnosis and Differential Diagnosis of Deafness, Arch. Otolaryng. **28**:663-675 (Nov.) 1938.

tones, both the nerve and the organ of Corti in the basal coil are atrophic. The author states that the cause of these two types of lesions is unknown.

[Comment: The excellent work of Bunch on traumatic nerve deafness reported in this review last year indicates that the sharply localized impairment of perception for high tones with atrophy of the organ of Corti and the nerve is the result of exposure of the ear to loud sounds, usually over a prolonged period.]

Shambaugh [26] reports a series of 9 cases of perception deafness with diplacusis. In 1 the condition was apparently the result of a hemorrhage into the labyrinth, the diplacusis being due, the author suggests, to fibrin or red cells adhering to the vibrating membrane in the cochlea. In 1 case perception deafness with diplacusis resulted from serous labyrinthitis complicating acute suppurative otitis media. In 7 cases the condition was characterized by fluctuating deafness, usually with repeated attacks of vertigo. The author suggests that in this group there may be inflammatory labyrinthitis without suppuration of the middle ear but due to a focus of infection. The similarity to iritis was pointed out.

Hereditary Nerve Deafness.—Again the German literature is filled with articles on the differential diagnosis of hereditary and acquired deaf-mutism, a matter of considerable importance because of the law requiring the sterilization of those with hereditary deafness. Eschweiler [27] studied the hearing remnants in deaf-mute children between 12 and 15 years of age. He concludes that sterilization can be advised only with a family history of deafness. He agrees with Langenbeck that asymmetric hearing remnants indicate acquired deafness and symmetric remnants hereditary deafness. Along with the asymmetric hearing remnants in acquired deafness there is often asymmetry of the vestibular reactions.

Steinberg [28] advances the view that sterilization should not be required in otosclerosis but that it should be permitted in the interest of the patient, since pregnancy accelerates the process. Moreover, an otosclerotic person should not marry a person with a family history of otosclerosis, nor should relatives with otosclerosis in their family marry.

Lüscher [29] studied 5 temporal bones from 3 patients with endemic deaf-mutism and found pathologic changes chiefly in the middle ear,

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26. Shambaugh, G. E., Jr.: Recent Advances in the Diagnosis and Treatment of Deafness, Ann. Otal., Rhin. & Laryng. **47**:636-648 (Sept.) 1938.

27. Eschweiler, H.: Hunderd Erbgutachten aus der Taubstummenanstalt Leipzig, Ztschr. f. Hals-, Nasen- u. Ohrenh. **43**:231-249, 1938.

28. Steinberg, G.: Ueber die Schwierigkeit der Diagnose und Beurteilung familiärer Schwerhörigkeit, Ztschr. f. Hals-, Nasen- u. Ohrenh. **43**:501-524, 1938.

29. Lüscher, E.: Drei Fälle von endemischer Taubstummsheit, Schweiz. med. Wchnschr. **68**:835-838 (July 16) 1938.

consisting of an increase in the periosteal layer over the promontory, narrowing the oval window slightly and almost completely closing the round window niche. The inner ear and ossicles were normal.

[Comment: If deaf-mutism results from blocking the round window niche, as it seemed to in these cases, then one would expect that the operation suggested by Hughson would further impair rather than improve the hearing.]

Traumatic Nerve Deafness.—Caroggio [30] observed 15 cases of injury to the inner ear after work in compressed air. In all cases the symptoms occurred after decompression, from a few minutes to about one hour after return to normal atmospheric pressure. The duration of the latent period has no relation to the degree of damage. The typical syndrome is severe vertigo accompanied by falling and a dimming of consciousness, tinnitus and deafness. Nystagmus is often horizontal-rotary and directed toward the normal or less affected ear. The acute stage lasts about one week. In favorable conditions the vertigo and imbalance disappear gradually. Deafness varies from simple hypoacusis to complete deafness, and in 50 per cent of cases recovery is only partial. Vestibular hyporeactivity is found with the deafness.

Molan,[31] on the basis of experimental and histologic studies, believes that noise produces damage chiefly to hearing by air conduction but that that by bone conduction cannot be entirely excluded. The most injurious tone is that heard best by the human ear, namely, 2300 vibrations. An irregular noise is much more injurious than a uniform noise.

Tanturri [32] found a localized loss of hearing for tones around c-5 in 55 of 1,500 patients examined. He suggests that quinine, syphilis, trauma to the skull, nicotine and cocaine may be responsible for this.

[Comment: Bunch has fairly conclusively demonstrated that these localized dips at c-5 are usually the result of acoustic trauma.]

Bunch [33] observed a man of 22 who noted marked deafness in one ear immediately after a firecracker exploded close by. Audiometric tests showed a pronounced loss of perception for tones above 512 vibrations in this ear, with a positive reaction to the Rinne test, decreased bone conduction and perception in the Weber test referred to the left ear. Two days later the hearing had nearly returned to normal, there being only a dip of 45 decibels at 4096 vibrations. [The author does not hazard a guess as to the pathologic nature of this temporary loss.]

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30. Caroggio, L.: Lesioni cocleovestibolari nella malattia dei cassoni, Atti Soc. ital. di laring. (pt. 2), 1937, pp. 214-215.

31. Molan, A.: Otopatie da rumori, Atti clin. otolaring., Torino, 1938, pp. 372-389.

32. Tanturri, V.: Osservazioni sull'ipoacusia lecunare, Rassegna ital. di otorino-laring. **12**:195-215, 1938.

33. Bunch, C. C.: Traumatic Deafness from the Explosion of a Firecracker: A Case Report, Ann Otol., Rhin. & Laryng. **47**:1092-1095 (Dec.) 1938.

Nerve Deafness from Drugs.—Considerable interest continues to be shown in the effect of quinine on the hearing. Covell [34] studied the endings of the cochlear nerve in the hair cells by dissecting the membranous labyrinth of guinea pigs and placing the fragments in a dilute solution of methylthionine chloride (methylene blue). The hair cells of animals that were given three injections of quinine over ten days showed swelling of the nerve ending in the cell, followed later by a granular break-up of these endings, with deeper than normal staining. Marked degenerative changes in the cytoplasm also were seen in many of the hair cells from the treated animals. Not all the hair cells of one cochlea showed the same degree of change, but the changes were, on the whole, more pronounced toward the lower half of the cochlea. The author suggests that these findings, which are not demonstrable in ordinary histologic preparations, may represent one of the earliest alterations in the neural mechanism for the transmission of impulses.

Covell [35] measured the area of the stria vascularis as a whole compared with the vascular areas alone in the midvertical section of the cochlea in guinea pigs given injections of quinine, sodium salicylate, codeine or soluble barbital daily for thirty-six to fifty-seven days. A significant increase in the vascular areas was found in the animals treated with quinine and with sodium salicylate; codeine resulted in a slight increase, while soluble barbital produced a very slight decrease, probably of no significance. The author suggests that the vascular stasis induced by quinine and salicylates results in deficient nourishment of cochlear structures and might alter the intracochlear fluid pressure. In addition to the possible impairment of nutrition from vascular stasis, these drugs probably also are direct protoplasmic poisons.

Covell [36] summarizes the results of several years of experimental study of the effects of quinine and salicylates on the cochlea. The vessels of the stria vascularis were found to be definitely dilated. The mitochondria of the cells of the stria vascularis revealed degenerative changes in pregnant guinea pigs and even greater changes in the fetal guinea pigs. The cells of the external sulcus were swollen and vacuolated but without mitochondric damage. Degenerative changes in the external hair cells, consisting of vacuolation, distortion and transformation of the fine rodlike mitochondria into droplets, were commonly found, the internal hair cells being more resistant. The myelin sheath

34. Covell, W. P.: The Peripheral Endings of the Cochlear Nerve, Ann. Otol., Rhin. & Laryng. **47**:63-67 (March) 1938.

35. Covell, W. P.: Histopathology of the Peripheral Auditory Mechanism in Drug-Injected Animals, Ann. Otol., Rhin. & Laryng. **47**:342-346 (June) 1938.

36. Covell, W. P.: Effects of Drugs on the Stria Vascularis, Arch. Otolaryng. **27**:438-443 (April) 1938.

of the cochlear nerve is exceptionally unstable, and degenerative changes in it are produced by a variety of factors, even control animals showing some changes but not as extensive as those in the drug-treated animals. Changes in the cells of the spiral ganglion, with vacuolation of the cytoplasm, loss of the chromophilic substance and tendency to displacement of the nucleus occurred with quinine and salicylates and were more severe after administration of salicylates.

The author concludes that quinine and salicylates act not alone on the vascular supply of the cochlea but on the entire structure. Any one of the many changes described might cause an impairment in hearing.

Mosher [37] gave an average of 20 grains (1.3 Gm.) of quinine to 11 pregnant guinea pigs in one to ninety-two doses and obtained from them 23 fetuses for study. Three pregnant guinea pigs were given sodium salicylate, from 20 to 35 grains (1.3 to 2.3 Gm.), and yielded 7 fetuses for study. One adult was given mapharsen, and its fetus was studied. Six adults were used as controls.

Histologic study of the temporal bones from these animals showed small hemorrhages into the cochlea, most often into the scala tympani of the basal whorl. Of 5 adult controls, 4 showed slight hemorrhages into the cochlea but, as a rule, the hemorrhages were more extensive after administration of quinine and were associated with marked congestion of the cochlear blood vessels. Mapharsen and sodium salicylate resulted in similar changes in the fetus and the adult except that mapharsen resulted in hemorrhages into the scala vestibuli, vestibule and semicircular canals rather than the scala tympani, suggesting that quinine and mapharsen may have a selective action on different parts of the inner ear.

Falbe-Hansen [38] gave 4 or 5 grains (0.26 or 0.32 Gm.) of salicylates a day to 41 patients, and 33 of these showed definite diminution of hearing, as well as vestibular symptoms and tinnitus. After stopping the medication complete recovery occurred. Quinine, 80 to 150 cg., was given to 21 patients, all of whom showed deafness, tinnitus and vertigo with nystagmus. Patients with aural disease were especially sensitive to quinine. The author points out the great similarity between these symptoms and Ménière's disease and suggests that in both instances the same cause is present, namely, edema of the labyrinth. By dehydration the author improved patients with Ménière's disease and by hydration he brought back the symptoms. He suggests that the loss of hearing from quinine and salicylates may be due to capillary injury and edema.

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37. Mosher, H. P.: Does Animal Experimentation Show Similar Changes in the Ear of Mother and Fetus After the Ingestion of Quinine by the Mother? Laryngoscope **48**:361-395 (June) 1938.

38. Falbe-Hansen, I.: Osservazioni cliniche e sperimentali sull'influenzae dei salicilati e del chinino sull'organo dell'udito, Rassegna ital. di oto-rino-laring. **12**:81-86, 1938.

Chryssicos and Yanoulis [39] studied 28 persons who had taken huge doses of quinine with suicidal intent (as high as 16 grains [1 Gm.]). Extraordinarily loud tinnitus was always present, and objectively there was perception deafness, which was never permanent. There were vertigo, nystagmus, falling and nausea. The galvanic and caloric reactions were always reduced. A new symptom also was observed in a series of such cases, namely, congestion of the drum membrane and in 1 case acute exacerbation of chronic otitis.

[Comment: It is surprising that there was no permanent defect in hearing after these huge doses of quinine. Perhaps prolonged medication is more apt to lead to permanent deafness than is one large dose.]

There is an interesting report from Farquharson [40] of deafness in a dog as a result of oil of chenopodium administered for worms. A total dose of 1.5 cc. in a capsule was given.

Chavanne [41] has observed 3 cases of transitory deafness after the injection of antityphoid vaccine. Three antidiphtheritic injections were given a child, and after each injection the child vomited, was feverish and complained of headache and defective hearing on the right side. Eight months later reexamination showed inner ear deafness on the right side. The author advises withholding further injections when such symptoms occur after vaccination.

Nerve Deafness from Infections.—A study of the hearing and vestibular apparatus in 115 children in the institute for the deaf and dumb in Leningrad and of 18 children hard of hearing from cerebrospinal meningitis was carried out by Bubes.[42] Loss of vestibular function was found in 91 per cent of all the cases, 81 per cent showing complete absence of response and 10 per cent diminution of response. On the other hand, complete deafness was found in only 31 per cent of the children in the institute. In the 18 children hard of hearing from cerebrospinal meningitis, the vestibular response was completely lost in 13, diminished in 3 and normal in 2. Especially noteworthy were 4 subjects with normal hearing in one ear and complete loss of vestibular function in both ears. The author concludes that in cerebrospinal meningitis the vestibular apparatus has a greater tendency to be damaged

39. Chryssicos, J., and Yanoulis, G.: L'influence de la quinine à hautes doses sur le labyrinthe, Presse méd. **46**:1877-1878 (Dec. 21) 1938.

40. Farquharson, J.: Deafness Due to Toxicity of Oil of Chenopodium, J. Am. Vet. M. A. **93**:329 (Nov.) 1938.

41. Chavanne, F.: Un cas de surdité unilatérale consécutive à la vaccination antidiphtérique, Oto-rhino-laryng. internat. **22**:481-482, 1938.

42. Bubes, G. F.: Zur Frage der Affektion des Gehörorgans bei der epidermischen cerebrospinalen Meningitis, Vestnik otol. **2**:217-218, 1938.

than the cochlear, resulting in the so-called paradoxic injury to the ear, in which the phylogenetically older vestibular apparatus is more damaged than the newer cochlea.

Toglia [43] discusses the different theories explaining the occurrence of deafness after mumps. Toxic labyrinthitis, suppurative labyrinthitis via the glaserian fissure and the middle ear and metastatic infection such as occurs in the ovaries, pancreas and testes have all been suggested. Lately the theory of meningoencephalitis due to a neurotropic toxin has gained ground. While meningeal symptoms in epidemic parotitis are relatively rare, there are always slight changes in the cerebrospinal fluid, indicating a meningeal or encephalitic reaction. Apparently the virus fixes directly in the central nervous system at the onset of the disease.

Loebell [44] reports a case of multiple recurrent osteomyelitis in which vertigo, vomiting and headaches developed and four weeks later defective hearing in one ear.

MacCready [45] describes the interesting case of a boy of 15 who was given a prophylactic dose of 3,000 units of tetanus antitoxin intramuscularly. Nine or ten days later he looked feverish; swelling of the right arm developed, and he was dizzy. Five days later these symptoms had subsided, but he awoke deaf. The hearing has remained stationary since then (one year). Examination shows inner ear deafness with a greater loss of hearing for high tones, the loss being 46 per cent in the right ear and 45 per cent in the left ear for the conversational tones. The author believes that the symptoms were the result of an anaphylactic reaction in both eighth nerves.

[Comment: Since anaphylaxis occurs only in certain animals and probably not at all in man, it would be more accurate to call this an allergic reaction of some sort, probably similar to serum disease, which may cause severe neuritis.]

Leicher [46] reports 5 cases of deafness following food poisoning. In the first the patient became deaf in the right ear with tinnitus and vertigo a few days after gastrointestinal symptoms caused by eating vanilla ice cream. The next 3 cases were those of a father, mother and son all ill with gastrointestinal symptoms after eating a meat salad with mayonnaise. In the son spontaneous nystagmus and bilateral nerve deafness developed; the father showed caloric hyperirritability and slight

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43. Toglia, C.: Sordità da parotite epidemica, Rinasc. med. **15**:655-656 (Oct. 15) 1938.

44. Loebell, H.: Sulla sordità da osteomielite, Rassegna ital. di oto-rino-laring. **12**:31-37, 1938.

45. MacCready, P. B.: Inner Ear Deafness from Tetanus Antitoxin Injection, Ann. Otol., Rhin. & Laring. **47**:247-252 (March) 1938.

46. Leicher, H.: Neuritis nervi octavi durch Nahrungsmittelvergiftungen, Hals-, Nasen- u. Ohrenarzt. (pt. 1) **29**:104-107 (Feb.) 1938.

transitory nerve deafness, while the mother showed disturbance of the right vestibule. In the fifth case gastrointestinal symptoms developed after the eating of ham. There were vertigo, headache, tinnitus on the left, deafness and absence of vestibular response. A year later the vestibular response on the left side had returned but this ear was still deaf.

[Comment: The organism causing the 3 attacks in the same family must have created a toxin with an affinity for the eighth nerve.]

König [47] studied a large series of patients to see how often defects of hearing following diphtheria, scarlet fever and measles are the result of toxic neuritis of the inner ear. Among 1,602 patients with diphtheria there were 117 with otitis, and 8 of these remained deaf because of a conduction defect. In no case could true toxic neuritis of the eighth nerve be demonstrated, although other neural disturbances, such as paralysis of the palate, larynx or ocular muscles, were fairly frequent. Among 995 patients with scarlet fever there were 177 with otitis, of whom 15 remained deaf. Among 671 patients with measles there were 140 with otitis, of whom 7 remained deaf. In each case deafness from measles or scarlet fever was of conduction type, and no case could be regarded as one of toxic neuritis. The author concludes that while toxic neuritis of the nerve of hearing may occur after diphtheria, scarlet fever or measles, it is certainly rare and is probably due to abnormal sensitivity of the nervous apparatus.

[Comment: These findings differ from the results of the statistical study in the schools for the deaf carried out several years ago, in which most of the acquired deafness appeared to be due to toxic neuritis from these infections without otitis media.]

Three articles report cases of syphilitic deafness. Carnevale Ricci [48] studied the ears of 43 patients with congenital syphilis and found marked changes in the ears characteristic of syphilis in 18 per cent. Syphilitic foci were found in all parts of the hearing organ in various degrees and stages of development. The author concludes that syphilitic involvement of the ear is more common in congenital syphilis than is generally appreciated.

Urechia [49] reports 2 cases of syphilitic labyrinthitis, in 1 of which the condition followed an injury to the skull. In the first, a man of 42 had acquired syphilis twenty years before and was treated only by a single series of injections. A heavy weight fell on his head, causing

47. König, R.: Schädigung des Gehörorgans bei Diphtherie, Scharlach und Masern, Ztschr. f. Hals-, Nasen- u. Ohrenh. **43**:250-266, 1938.

48. Carnevale Ricci, F.: Osservazione istopatologische sulla sifilide congenita dell'orecchio, Arch. ital. di otol. **50**:521-634 (Oct.) 1938.

49. Urechia, C. I.: Paralysie générale traumatique et labyrinthite: Tabes conjugal et labyrinthite, Paris méd. **2**:210-211 (Sept. 24) 1938.

unconsciousness for ten minutes. Five months later he complained of rheumatism in his legs and was found to have dementia paralytica with bilateral neurolabyrinthitis and slight deafness. Wassermann tests of both the blood and the spinal fluid were positive. The labyrinthine symptoms disappeared after antisyphilitic and malarial treatments, but the defect in hearing remained. In the second case, a man of 55 had acquired syphilis twenty-two years before and was not treated. He complained of slight dizziness and loss of hearing. He was found to have tabes with bilateral deafness; Wassermann tests were negative for the blood but positive for the spinal fluid. The symptoms completely cleared up under malarial and antisyphilitic treatment. The author states that bilateral labyrinthitis with tabes is rare but might be seen more often if all tabetic patients were systematically investigated.

Ruskin and Hyslop [50] report a case of syphilitic deafness preceding the appearance of the chancre. A man of 40 was seen a few hours after a blow on the head because of pain in the right ear, headache, vertigo and nausea. The hearing in the right ear seemed diminished. Two weeks later the loss of hearing was greater, and a few weeks after this second examination a pimple on the glans penis was first noted. This was definitely diagnosed as a chancre. Four months later, in spite of antisyphilitic treatment, the right ear showed complete deafness. The authors base their diagnosis of syphilis antedating the chancre on the fact that when the patient was first seen the caloric test produced no reaction on the right side but the rotation test produced an equal response on each side.

[Comment: It is difficult to accept this as a case of syphilis of the inner ear preceding the chancre purely on the basis of paradoxic labyrinthine tests.]

Nerve Deafness from Allergy.—The importance of allergy in the middle and the inner ear is suggested by Jones,[51] who reports 3 cases. In a child of 7 known to be sensitive to chocolate, wheat and nuts earache and an injected drum membrane developed after the eating of nuts, involving first one ear and then the other, with both returning to normal two days later. A farmer complained of attacks of vertigo one or more times a week for a year and a half. Because he was a great drinker of milk he was placed on a milk-free diet and has remained free from attacks. A boy of 8 had almost continuous colds and attacks of otitis media, with malnutrition, constipation and diarrhea. He was placed on a diet excluding the common sources of allergy, namely, milk,

50. Ruskin, S. L., and Hyslop, G. H.: Acute Syphilis of the Internal Ear: An Interesting Case Report, Laryngoscope **48**:280-285 (April) 1938.

51. Jones, M. F.: Manifestations of Allergy in the Ear, Ann. Otol., Rhin. & Laryng. **47**:910-916 (Dec.) 1938.

eggs, chocolate, wheat and shell fish, and was placed in an environment free from the common allergenic inhalants. Improvement was rapid and steady.

[Comment: The author's reports are suggestive but not conclusive. To prove an allergic cause the symptoms must clear up on the removal of a substance and then be reproduced by adding the substance.]

Nerve Deafness from Miscellaneous Causes.—Jerlang and Dederding [52] made the surprising observation that of 28 patients submitted to intracranial section of the fifth nerve, 26 showed diminution in hearing several days after operation. There was marked impairment of perception for low tones, the upper limit being lowered in only 2 cases. The ear on the side not operated on was similarly involved, to a lesser degree. Tinnitus was present in 11; spontaneous nystagmus, in 20. Several months later the hearing was normal in some, improved in some and unchanged in some. The authors suggest vasomotor labyrinthine edema as a result of the sympathetic nerve connections between the carotid plexus, the middle ear and the gasserian ganglion, through the circle of Willis to the opposite side.

[Comment: The clinical observation is interesting but the explanation for it is rather involved.]

Yoshimura [53] studied 14 tumors of the cerebellopontile angle and concludes that dizziness is to be regarded as an early symptom in addition to perception deafness. The labyrinthine tests show diminution or absence of the response to rotatory, caloric or faradic stimulation. There may be spontaneous nystagmus of the first degree to both sides, which is slower and with a wider amplitude on looking toward the side of the tumor. There are vertical nystagmus on looking up and ataxia with deviation toward the side of the tumor. Involvement of the third, fifth, sixth and seventh nerves is observed. The roentgenogram shows a widening of the internal acoustic meatus with destruction of the pyramid.

Clark and Russell [54] describe the histologic findings in a patient with cortical deafness. A woman of 44 suffered a stroke with paralysis of the left arm and leg, from which a partial recovery was made. The hearing was normal. Two months after the initial attack a second stroke occurred. Examination revealed complete bilateral deafness with normal response to the caloric test in each ear and no definite signs of weakness

52. Jerlang, E., and Dederding, D.: Akustische und vestibuläre Funktionsstörungen nach temporaler retro-ganglionärer Trigeminotomie, Acta oto-laryng. **26**:625-631, 1938.

53. Yoshimura, M.: Beiträge zur Kenntnis der Gehörorganstörungen bei Kleinhirnbrückenwinkeltumoren, Ausz. z. Otol. (Tokyo) 32-36, 1937, pp. 32-36.

54. Clark, W. E. L., and Russell, W. R.: Cortical Deafness Without Aphasia, Brain **61**:375-383 (Dec.) 1938.

or anesthesia, the previous hemiplegia having practically cleared up. There was no aphasia, and the patient was soon able to be up and around, complaining only that she could not feel the ground with her feet. The hearing did not return. Six months later death occurred from a pontile hemorrhage. Postmortem examination revealed an old hemorrhage into each external capsule cutting off the fibers to the upper portion of the temporal lobes of either side. The authors call attention to the fact that while a total of 18 cases of cortical deafness with postmortem examination have been reported, this is the first time that deafness was present without aphasia.

[Comment: Because of the bilateral cortical representation of the auditory tracts from each ear a lesion of one temporal lobe does not result in loss of hearing, which has led some to doubt that the center for hearing is in the temporal lobes. The unique case reported by Clark and Russell is definite proof that the center of hearing is located in the upper portion of the temporal lobes on both sides.]

Döderlein [55] suggests logically that the better the function of an organ is the closer it is to normal and that true presbycusis can be studied only in old people with relatively good hearing. He accordingly studied the oldest people with the best hearing that he could find, a total of 50 between the ages of 80 and 94 being considered suitable. The lower tone limit was determined with difficulty and lay around 64 vibrations. The upper tone limit was easily obtained and lay around 4096 vibrations or a little higher. Between the ages of 20 and 50 the upper tone limit falls from 20,000 to 15,000 vibrations, and between 50 and 80 it falls from 15,000 to about 5,000 vibrations. The hearing for the voice also declines from perception at 15 meters at 20 to perception at 4 meters or less at 80, representing a loss of about three fourths of the hearing. If the loss of hearing is greater than this it must be regarded as pathologic. The histologic changes heretofore described for presbycusis have been based on ears of deafened people and, therefore, are to be regarded as pathologic rather than physiologic. To determine the histologic changes of presbycusis it will be necessary to study the ears of old people with perception of a whisper at up to 4 meters and an upper tone limit up to 5,000 vibrations. The author agrees with O. Mayer that changes will be found to occur in the elastic tissue of the basilar membrane similar to the loss of elasticity in the lens of the eye. Since perception for the highest tones is used least, the elasticity as it affects this is lost first.

Nussdorfer [56] studied audiometrically 300 patients between the ages of 20 and 70 and found a progressive lowering of the upper tone limit

55. Döderlein, W.: Ueber Presbyacusis, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **144**:295 (April) 1938.

56. Nussdorfer, R.: Grafici audiometrici dell'orecchio senile, Atti clin. otolaring., Torino, 1938, pp. 437-453.

with a progressive elevation of the threshold over the entire tonal range and with relatively slight elevation of the lower tone limit. There was also abnormal fatigability of the senile ear for frequencies above 512 and a sudden dip to 4096 vibrations in a large proportion of cases.

Torrini [57] fed 6 guinea pigs a diet free from vitamin B and 6 a diet free from vitamin C, and after the animals had died as a result of the diet the inner ear was examined. The greatest changes were in the labyrinthine capsule, with early osteoporosis and with some halisteresis in those deprived of vitamin C. The organ of Corti showed no changes. The cells of the spinal ganglion showed degeneration with formation of vacuoles.

Kobrak [58] suggests that primary nerve deafness may result in a secondary reflex anomaly of the muscles of the middle ear, which in turn may further impair the function of the inner ear, thus resulting in a vicious circle. This, he suggests, would account for the undeniable benefit of inflation in many cases of inner ear deafness.

Treatment of Nerve Deafness.—A few new treatments for nerve deafness were reported in 1938, none of them especially convincing or logical. Koch [59] reports the results of the treatment of the deafness of old age with various glandular preparations given by intramuscular injection. Of 19 patients treated, 7 complained of marked tinnitus without deafness and 6 were relieved. Twelve who were deafened each experienced improved hearing.

[Comment: There is no reason to expect that the purely degenerative and atrophic changes of presbycusis can be altered by any form of treatment. Results of this sort are of no value unless controlled by a parallel series of patients receiving no treatment.]

An experimental study of the effects of radiation on the ear was carried out on guinea pigs by Doi [60] with one or two needles of 5.5 mg. of radium bromide placed for twenty-four hours to twenty-five days in the middle ear. It was found that continuous irradiation with small doses produced primary degeneration of all the endings in the labyrinth, first in Corti's organ, then in the ganglion cells and lastly in the nerve fibers, with more damage in the cochlear than in the vestibular apparatus.

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57. Torrini, G.: Avitaminosi ed orecchio interno ricerche sperimentali, Arch. ital. di otol. **50**:658-672 (Nov.) 1938.

58. Kobrak, F.: Pathologische Physiologie und praktische Ohrenheilkunde: II. Die Mittelohrinsuffizienz, eine funktionelle Miterkrankung des Mittelohrs, Pract. oto-rhino-laryng. **1**:293-300, 1938.

59. Koch, F. X.: Die Hormonbehandlung der Altersschwerhörigkeit, Monatschr. f. Ohrenh. **72**:777-790 (Aug.) 1938.

60. Dei, S. I.: Experimentelle Studien über den Einfluss der Radiumbestrahlung auf den Gehörorgan, Okayama-Igakkai-Zasshi **50**:1845-1846 (Sept.) 1938.

The greater the immediate dose with the same complete dose, the greater the degeneration of the labyrinth. No changes were found in the non-irradiated ear.

Further evidence that roentgen rays cannot have a beneficial effect on the ear directly is an article by Hays,[61] who emphasizes that otosclerosis or nerve deafness cannot be affected by roentgen treatment. On the other hand, when the deafness is the result of occlusion of the eustachian tube by hypertrophy of lymphoid tissue in the nasopharynx or eustachian tube, roentgen treatment can be of benefit.

Kupfer[62] theorizes that the Wever-Bray response is due to the solids and liquids in the cochlea acting as variable condensers and dielectric solutions. Movement of this fluid (by the footplate of the stapes) sets up electric currents. On the assumption that tinnitus and deafness may be due to electric currents in the diseased organ the author led into the ear electric currents from outside to neutralize these pathologic currents and improve the hearing. He claims good results in many cases.

[Comment: This is another bizarre form of treatment based on a nebulous theory.]

An even more fantastic type of treatment is advocated by Zajicek,[63] who reports on 40 patients with conditions such as inner ear deafness, presbycusis and adhesive middle ear deafness treated with "hormones" (not specified) plus the application of a "respiratory ferment ointment" (*Atmungsfermentsalben*) once a week behind the ear and over the adjacent part of the head and neck for four or five times. At the first application the patients declared that they could notice deep warmth, with a clearer feeling in the ear.

[Comment: Should scientific journals publish such manifestly unscientific material?]

Kanizsai[64] exposes the ears of deaf-mutes to loud sounds of from 25 to 1200 vibrations. A soon as there is a reaction, the sound is decreased until the threshold is reached. The purpose is to obtain a response from Corti's organ over as large a range as possible with the slightest possible stimulus. The author emphasizes the inestimable

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61. Hays, H.: The Truth About the X-Ray Treatment of Deafness, Laryngoscope **48**:176-182 (March) 1938.

62. Kupfer, E.: On the Origin of the Wever-Bray Response and on an Electrotherapy of the Ear, J. Laryng. & Otol. **53**:16-31 (Jan.) 1938.

63. Zajicek, O.: Die Hormonbehandlung der progressiven Schwerhörigkeit mit Atmungsfermentsalben als aussichtsreicher Weg, Wien. med. Wchnschr. **88**: 213-215 (Feb. 19) 1938.

64. Kanizsai, D.: Ueber mein heiltechnisches Verfahren bei der Gehörentwicklung bei Taubstummen und Personen mit Hörresten, Monatschr. f. Ohrenh. **72**:277-287 (March) 1938.

psychologic significance of even small results in awakening the hearing of the tones for which perception remains.

[Comment: It is difficult to conceive of any benefits other than psychologic of this or of any other treatment of the hearing of deaf-mutes.]

Ménière's Syndrome.—The many interesting articles on Ménière's syndrome that appeared in 1938 included contributions on the pathology, on the etiology and on the treatment of this dramatic disease.

Hallpike and Cairns [65] contribute an important article on the pathology of Ménière's syndrome. Except for a case of neurofibroma in the basal coil of the cochlea described by Wittmaack and a case of generalized encephalitis of traumatic origin described by Videbeck, there had been no histologic studies in cases of Ménière's syndrome, and the two previous reports were obviously of rare and unusual conditions and not of the ordinary attack of Ménière's syndrome. It may, therefore, be said that this study of Hallpike and Cairns of 2 patients who died after section of the eighth nerve for relief of vertigo is the first histologic study of Ménière's disease to be reported.

In the first case, a man of 63 complained of recurring attacks of vertigo for three years, increasing in severity until he lost his job. For one year there had been progressive deafness in the left ear. Hearing tests revealed diminished hearing in the left ear, with shortened perception in the Schwabach test and a positive reaction to the Rinne test. The caloric test showed no response from this ear but a prompt response from the right. Death occurred three days after section of the eighth nerve, and autopsy was done six hours after death. Both drum membranes were thickened, with bony thickening of the posterior edge of the left footplate of the stapes apparently of inflammatory origin. The left saccule and scala media were grossly dilated, with obliteration of the perilymph spaces of the vestibule and scala vestibuli. Corti's organ was degenerated on the left side. The normal perisaccular connective tissue was absent on both sides. The most striking change was the displacement of Reissner's membrane so that it lay flattened against the roof of the scala vestibuli with organizing connective tissue holding it in this position.

In case 2 a man of 28 had increasing deafness in the left ear and vertigo for four years. Hearing tests revealed defective hearing in the left ear, air conduction being better than bone conduction. There was an equal response to caloric stimulus from both sides. Death occurred the day after section of the eighth nerve, and autopsy was performed four hours later. The left temporal bone only was examined histologi-

65. Hallpike, G. S., and Cairns, H.: Observations on the Pathology of Ménière's Syndrome, J. Laryng. & Otol. **53**:625-655 (Oct.) 1938.

cally. Again there was gross dilatation of the saccule and scala media, with obliteration of the perilymphatic cistern and the scala vestibuli. There was degeneration and rupture of the wall of the anterior vertical membranous canal with a dense albuminoid coagulum in its lumen. Corti's organ was degenerated, and the epithelium of the maculas and of the cristae of the canals was degenerated. The stria vascularis was degenerated. The normal area of perisaccular connective tissue around the saccus endolymphaticus was absent. Again the displacement of Reissner's membrane to the wall of the scala vestibuli was most striking.

In discussing these findings the authors point out that while the changes are consistent with serous labyrinthitis, the extreme and uniform dilatation of the endolymphatic system is extremely rare in serous labyrinthitis, nor can it be ascribed to the operation, since 5 patients who died after removal of acoustic tumors failed to show more than an insignificant displacement of Reissner's membrane. The authors suggest that in Ménière's syndrome there is increased endolymphatic pressure due to increased secretion or diminished absorption of endolymph. The bony spaces are finally filled by the increased endolymph until relatively slight increases in pressure result in anoxemia of the labyrinth and unopposed action of the opposite labyrinth. Finally, the authors suggest that diplacusis might occur because of stretching of the basilar membrane.

[Comment: The essential pathologic change, namely, extreme dilatation of the endolymph space must be regarded as the end result of the condition. These patients died between attacks, not during an attack. The pathologic picture during an attack remains to be described.]

An excellent description of Ménière's disease, based on a study of 117 patients seen during the past nine years, comes from Crowe.[66] Of the total number the vestibular nerve was divided for 94, while therapy was not given to 23 with mild attacks. The disease was unilateral in 90 per cent, with marked impairment of hearing in the affected ear. The first symptom appeared between the ages of 30 and 60 in 80 per cent. Only 10 of 71 patients had a history of otitis media at any time, and only 5 had a history of allergy or migraine. A positive Wassermann reaction was obtained from only 2.

The vestibular responses (minimal caloric reaction) were normal in 35 per cent, subnormal in 19 per cent and absent in 29 per cent, while in 17 per cent the tests were not made. Some patients with no caloric response in the affected ear had vertigo and were relieved by section of the vestibular nerve. The vestibular tests, therefore, have no value in the diagnosis and prognosis of Ménière's disease.

The deafness was of the inner ear type. It was usually progressive but had the peculiar characteristic of fluctuating. In this respect the

66. Crowe, S. J.: Ménière's Disease, Medicine **17**:1-36 (Feb.) 1938.

deafness differs from inner ear deafness due to all other causes. The tinnitus likewise fluctuates.

The vertigo occurs characteristically in attacks, with periods of complete freedom from dizziness between attacks. However, in some cases there is constant dizziness, punctuated by sudden severe attacks of vertigo. Spontaneous remissions are frequent and vary from a few months to twelve years. Because of these remissions one must be cautious about judging the effects of therapy.

The diagnosis of Ménière's disease is made by the history of attacks of vertigo. Deafness and tinnitus are usually, but not necessarily, present and may precede the onset of vertigo. Loss of consciousness may occur during the attacks of dizziness but never with convulsions.

The treatment of Ménière's disease is section of the vestibular portion of the eighth nerve and always results in cure of the vertigo. When the deafness is bilateral one vestibular nerve is cut, and if the vertigo persists, the other nerve is cut. Of 72 cases the hearing after operation was worse in 22, unchanged in 36 and improved in 14.

The cause for Ménière's disease is unknown. The author believes, however, that it lies in the labyrinth and may be a disturbance of the chemical composition or pressure of the endolymph.

[Comment: This authoritative description of Ménière's disease is based on an unusually large number of cases. Many will disagree with the author's statement that surgical section of the vestibular nerve is the only treatment. In my own experience, allergy or focal infection accounts for the condition in most cases, and removal of the food or foods to which the patient is allergic results in complete and immediate relief from the vertigo and tinnitus and usually in improvement in the hearing. Cutaneous tests are of only occasional value, since they are negative in many cases. One must employ an elimination diet, eliminating wheat, milk, eggs, chocolate, coffee, citrus fruits and other common causes of food allergy, and then add these foods one by one until the offending substance is discovered.

The fluctuating nature of the deafness in Ménière's disease, brought out by Crowe, is confirmed by my own observations. In addition, the patients all, or nearly all, show diplacusis.]

Mygind and Dederding [67] continue their writing on Ménière's disease as a manifestation in the ear of disturbed water metabolism. They

67. Mygind, S. H., and Dederding, D.: Ménière's Disease as an Indication of Disturbances in Water Metabolism, Capillary Function and Body Condition, Ann. Otol., Rhin. & Laryng. **47**:55-62 (March) 1938; Clinical Experiments with Reference to the Influence of the Water Metabolism on the Ear, ibid. **47**:360-369 (June) 1938; The Diagnosis and Treatment of Ménière's Disease, ibid. **47**:768-774 (Sept.) 1938; The Pathogenesis of Ménière's Disease and of Kindred Conditions in the Ear and the Rest of the Body, ibid. **47**:938-946 (Dec.) 1938; The Problems of Aural Medicine, J. Laryng. & Otol. **53**:35-46 (Jan.) 1938.

define Ménière's disease as an aural disease with periodically varying acoustic and vestibular symptoms which do not present any special cause (such as symptoms referable to the middle ear, syphilis, tumor of the brain and trauma). The hearing is nearly always reduced, the tests always revealing a disturbance of the sound-conducting mechanism with elevation of the lower tone limit as well as loss of perception for upper tones by air conduction (but not by bone conduction). The bone conduction for a-1 (435 vibrations) may be shortened, but this is consistent with disturbance of the sound-conducting mechanism rather than perception deafness as previously believed.

[Comment: I cannot agree with these authors that shortened bone conduction is consistent with conduction deafness and that the patients have conduction rather than perception deafness. True, they show a loss of perception for low tones as well as for high, but, as I have pointed out, this is low tone perception deafness and not conduction deafness.]

The most characteristic feature of the hearing is its fluctuation, with changes from day to day or even from hour to hour. The tinnitus also is variable in intensity and is not always present. Autophony and diplacusis are not rare.

Spontaneous nystagmus is always present at some time, and the oftener the patient is examined the more frequently it will be seen. The direction of the nystagmus can be toward or away from the affected ear. The vertigo occurs in all forms and degrees.

In addition to the aural symptoms, certain general symptoms are common in such patients. Headache is the most frequent and may be in the form of migraine. Subcutaneous nonpitting edema is frequent. Vasomotor rhinitis, gastrointestinal disturbances and rheumatism are other frequent symptoms. Variations in the quantity and weight of urine are not uncommon, retention of water often preceding an attack of vertigo.

Perspiration from physical exercise may give relief from the dizziness, while the ingestion of much fluid does not agree with the patients. A good many have of their own accord reduced their consumption of liquid and salt.

Clinical experiments on water metabolism in Ménière's disease were carried out by the authors. Mersalyl, a powerful diuretic, was injected intravenously (1 cc. of a 10 per cent solution) seventy-seven times for 46 patients, and in 33 a definite improvement in hearing resulted, usually toward the end of the diuresis. The lower tone limit improved as much as from 150 to 55 vibrations, and perception of the whispered voice from at the ear to 20 meters. The opposite experiment was carried out, and 1,000 cc. of water was administered to the fasting patient before breakfast. A great number of patients with Ménière's disease showed more

or less pronounced reduction in hearing during the following four hours, with elevation of the lower tone limit and shortening of air and bone conduction for a-1 (435 vibrations), as well as impairment of perception for the whisper. The results were the same as those obtained by Furstenberg. The beneficial effect of the Furstenberg diet is due, the authors believe, to the fact that it is dehydrating, since it is poor in salt, rather than to its restriction of the intake of sodium.

In the treatment of Ménière's disease the other conditions that may produce the same symptoms must first be ruled out, namely, suppuration of the middle ear, tubal catarrh and stenosis, syphilitic neurolabyrinthitis, otosclerosis, epidemic encephalitis, disseminated sclerosis, cerebral tumor and tumor of the acoustic nerve.

The treatment of an acute attack of Ménière's disease consists of rest in bed in a dark room with a minimum of medication. For severe attacks 0.5 mg. of atropine given subcutaneously has seemed to be of benefit. Aside from reducing the intake of fluid and salt, nothing more is done for the acute attack. Before beginning even this treatment it is helpful to have the patient record all that he eats and drinks for three or four days of his usual diet. He is then submitted to the water retention test. Before breakfast he is weighed and empties his bladder. He then drinks 1,000 cc. of water, and after four hours he is weighed and his total diuresis is measured. If he retains 250 to 300 cc., his intake of fluid, including drink, vegetables and fruit is limited to 600 cc. If the retention of water is greater, the intake of fluid may be limited to 500 cc. or even 400 cc., while if there is no retention he is allowed 800 cc. of liquid. Food may be prepared as usual, but he is not allowed to add any salt, and salty foods are avoided.

Besides the diet, ultraviolet light baths are given to increase the cutaneous circulation, and baths, massage and exercise may be used to improve the vasomotor tone. Daily catheterization of the eustachian tube prevents stenosis, mobilizes the ossicles and influences the content of the labyrinth via the fenestrae. It is desirable to hospitalize the patient during the first month in order to acquaint him thoroughly with his disease, to provide sufficient rest and quiet and to regulate his regimen strictly. After three to six months of the regimen at home most patients may resume an ordinary mode of diving, but subject to recurrences after infections, overexertion, pregnancy and the menopause. As a rule, the dizziness will be cured, or at least reduced to a minimum. Of those followed for three years or more 81 per cent remained permanently free from vertigo. The hearing was distinctly improved on dismissal from the hospital in more than half and about half of those (one fourth of the total) maintained their improvement. In isolated cases the condition defies all treatment.

As a result of their observations, the authors conclude that in Ménière's disease there is disturbance of water metabolism, not only in the ear but also in other parts of the body. The periodic fluctuation of this disturbance is due to vasomotor dysfunction. Water is deposited in the labyrinth, eustachian tube, brain, nasal mucosa, gastrointestinal mucosa, muscles or subcutaneous tissues, according to the local disturbances in vasomotor reflexes. These accumulations of fluid are intracellular rather than intercellular, as evidenced by the failure of the subcutaneous edema to pit on pressure. As the process continues, the edema gradually changes into atrophic and fibrous alterations.

The retention of water in the labyrinth results in increased endolymphatic pressure and causes low tone deafness by fixation of the stapes from the inside. The diminished bone conduction in this conduction deafness is due to outward dislocation of the footplate of the stapes resulting in decreased contact between the stapes and the oval window. The vestibular symptoms are due to the intracellular pressure of the edematous sensory cells exceeding the endolymphatic pressure, or vice versa. The apoplectiform attacks are possibly due to sudden jerky yielding of the footplate of the stapes to the fluctuating endolymphatic pressure. Finally, the authors assert that there is no sharp boundary between typical Ménière's disease and common catarrh of the middle ear with tubal stenosis and neurolabyrinthitis.

[Comment: The authors' observations are interesting. It is remarkable that they have not thought of allergy as an explanation for the various edemas observed. A food allergy will explain better than anything else all the observations made by these authors.]

Quix,[68] in discussing the etiology of Ménière's disease, suggests that the fact that the vestibular apparatus usually recovers following the attack while the hearing does not is best explained by the fact that the attack occurs in the labyrinth and the cells of Corti's organ are nourished by the endolymph and are, therefore, more susceptible to endolymphatic changes, whereas the cells of the cristae and maculas are supplied by their own blood vessels. The author, however, is not sure but that Ménière's disease may be central rather than peripheral because of the headache, nausea and changes in corneal sensitivity on the same side.

Granström and Nylén [69] describe the case of a woman of 56 who for a number of years had suffered from attacks of vertigo and of iritis with

68. Quix, F. H.: La maladie de Ménière, Ann. d'oto-laryng., July 1938, pp. 596-604.

69. Granström, K. O., and Nylén, C. O.: Crises de vertige de Ménière et crises d'iritis avec oedème de Quincke dans les paupières chez une même patiente, Acta oto-laryng. **26**:717-725, 1938.

palpebral Quincke edema. They suggest that an allergic disorder was responsible for her symptoms, although it was impossible to determine the etiologic factor, the cutaneous tests all being negative.

[Comment: The authors made the common mistake of relying on cutaneous tests to determine food allergy. Since in many cases of food allergy, perhaps in the majority, the cutaneous tests are negative, one must use the elimination method for diagnosis.]

Alföldy [70] refers to the symptom complex of Ménière as "eighth nerve crises." He points out that the attacks are related to vasomotor rhinitis, asthma, Quincke's edema, mucous colitis and migraine. Nonspecific histamine therapy is used, at first intracutaneously to lessen the shock and observe the local reaction, and then subcutaneously in increasing doses every second day. In general, ten to twenty injections result in complete recovery.

[Comment: The allergic factor in Ménière's disease is again suggested by this contribution.]

Güttich [71] describes 2 cases of Ménière's syndrome following intense exposure to sunshine. In 1 there was a marked inner ear defect in hearing, and in both positional nystagmus was present. The symptoms had completely disappeared the next day in 1, but in the other the nystagmus lasted three weeks and permanent nerve deafness in one ear remained. The author suggests histamine poisoning of the central vestibular nuclei.

[Comment: The deafness indicates a peripheral rather than a central disturbance.]

Brain [72] calls attention to the fact that dizziness is a frequent symptom of anxiety neurosis and is usually associated with severe feelings of anxiety and symptoms of overactivity of the sympathetic nervous system. Vertigo is not unknown in petit mal, in migraine and with localized cortical lesions and may, in fact, occur with a tumor of the brain in any location. Vertigo may be produced by paralysis of the ocular muscle with diplopia. Cerebellar lesions may, but do not always, cause vertigo. Lesions of the brain stem, particularly disseminated sclerosis, cause vertigo, usually without deafness and tinnitus and with weakness of the oculomotor or facial nerve or other evidence of disseminated sclerosis. Finally, lesions of the eighth nerve, such as a tumor or an anomalous vessel, may cause vertigo.

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70. Alföldy, E.: Le traitement des "crises octavus" angioneurotiques selon les principes les plus modernes, Ann. d'oto-laryng., December 1938, pp. 1159-1163.

71. Güttich, A.: Ueber Ménièresche Symptome nach Insolation, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **145**:499-500, 1938.

72. Brain, W. R.: Vertigo: Its Neurological, Otological, Circulatory and Surgical Aspects, Brit. M. J. **2**:605-608 (Sept. 17) 1938.

Aural vertigo may be the result of acute labyrinthitis secondary to suppurative otitis media. More often it is part of Ménière's syndrome. This syndrome is characterized by the sudden onset in a previously healthy auditory apparatus of tinnitus, deafness and vertigo with ataxia, nausea, vomiting and fainting. The deafness is progressive, and there may be transitory loss of vision or diplopia during the attacks.

The age at occurrence averages 49. The most important etiologic factor is focal infection, the imbalance of water emphasized by Mygind, Dederding and Furstenberg being probably a contributory factor but not the main cause. Treatment consists of the eradication of any source of infection. Tobacco may be the cause in susceptible persons. Restriction of fluid may be helpful. Phenobarbital, ½ grain (0.03 Gm.) two or three times a day, is valuable. Surgical intervention is indicated only for the rare resistant condition.

Wright [73] emphasizes the role of focal infection in Ménière's syndrome. He states that labyrinthine vertigo may be recognized by the coincidence of tinnitus and deafness, though, rarely, these may be absent in the early stages. In addition, certain tones may be heard at a higher or lower pitch in the affected than in the normal ear, and the sounds may be actually painful.

[Comment: Since testing routinely for diplacusis I have found this symptom invariably present in Ménière's disease.]

The following conditions can cause labyrinthine vertigo: localized or diffuse suppurative labyrinthitis secondary to suppurative otitis media; mumps (usually unilateral); herpes of the geniculate ganglion; congenital syphilis resulting in a completely deaf ear, usually bilateral; acquired syphilis, usually in the late secondary stage with acute labyrinthine involvement resulting in complete destruction of the labyrinth in two or three weeks; fracture through the base of the skull and labyrinth; quinine, salicylates and tobacco, and focal labyrinthitis (Ménière's disease).

In the author's series of 70 cases of Ménière's disease or, as he prefers to call it, focal labyrinthitis, the average age was 47. In no case, on critical examination, was a pathologic change in the eustachian tube found. A focus of infection can be found in every case.

[Comment: Because tonsils are removed only exceptionally in England and because dental hygiene in England, as a rule, is poorer than in this country foci may be found more often there than in the United States. Certainly many patients with Ménière's disease seen in this country have no obvious foci of infection.]

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73. Wright, A. J.: Aural Vertigo, J. Laryng. & Otol. **53**:97-112 (Feb.) 1938; Labyrinthine Giddiness: Its Nature and Treatment, Brit. M. J. **1**:668-670 (March 26) 1938.

In 23 of the author's cases all foci were removed. In 10 the vertigo and tinnitus ceased and the hearing returned to normal. In 3 the vertigo ceased and the hearing improved, while in 8 the vertigo ceased but the deafness persisted. In 2 cases one focus was removed without improvement but other foci (teeth or tonsils) remained and are probably responsible. On the other hand, the author has observed no case of spontaneous cure with restoration of normal hearing, although the frequent long periods of freedom from vertigo make it difficult to evaluate the results of treatment.

The author believes that the condition in his 70 cases is a definite disease, consisting of a primary lesion of the labyrinth not resulting from a lesion of the middle ear. The clinical course is similar to that in chronic iritis with the difference that in iritis one can visualize the lesion and see that it is inflammatory.

[Comment: The similarity between iritis and certain cases of Ménière's disease has been pointed out by me also.]

Mackenzie [74] uses the term "neurolabyrinthitis" to designate any inflammation involving the eighth nerve and labyrinth which may be caused by syphilis, acute infectious fevers, chronic infections, poisons, exposure to cold or focal infection.

Syphilis affects the ear in four ways: 1. In acquired syphilis there is bilateral neurolabyrinthitis with perceptive deafness, vertigo, nystagmus at first toward and later away from the affected ear, hyperirritability of the labyrinth at first and later hypoirritability, strongly positive Wassermann reactions and other evidences of syphilis, diminished galvanic response and a favorable response to antisyphilitic treatment. 2. In congenital syphilis the onset is sudden and bilateral, with profound perception deafness, rotary vertigo and spontaneous nystagmus with early hyperirritability and late hypoirritability to the caloric test but normal galvanic response, mildly positive Wassermann reactions, other signs of congenital syphilis (teeth and eyes) and unfavorable response to treatment. 3. In gummatous infiltration of the floor of the skull and the temporal bone there are unilateral gradual perceptive deafness, vertigo and nystagmus, early hyperirritability and late hypoirritability to the caloric tests, strongly positive Wassermann reactions, involvement of the seventh, ninth or other cranial nerve and favorable response to early and intensive treatment with iodides. 4. In primary retrolabyrinthine atrophy from metasyphilis there is bilateral progressive perceptive deafness with only occasional mild vertigo and nystagmus, diminished caloric and galvanic response, frequent involvement of the posterior column of the spinal cord and mildly positive Wassermann reactions.

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74. Mackenzie, G. W.: Neurolabyrinthitis, Internat. Clin. **2**:126-134 (June) 1938.

Neuritis of the eighth nerve from acute infectious fever, especially mumps and meningococcic meningitis, is typically bilateral [a misstatement by the author, since deafness from mumps is typically unilateral], while neuritis from chronic infections (tuberculosis, leukemia and leprosy) and from poisons (snake venom, alcohol, salicylates, quinine, arsenic, mercury and lead) is usually bilateral.

Labyrinthitis from focal infection is by far the most frequent cause for Ménière's syndrome. There is practically always an attack of influenza some weeks or months before the onset. An abscessed tooth has been the most frequent focus in the author's experience. After removal of a focus the symptoms are relieved and the hearing is improved.

Leichsenring [75] treats acute attacks of Ménière's disease by prescribing a fast of several hours and then giving ½ to ¾ liter of linden blossom tea followed by hot packs and the injection of 0.01 to 0.02 of pilocarpine. After two hours the patient is rubbed down with French brandy and the bed is changed. If necessary the dose of pilocarpine is increased the following day. The treatment continues for several days. Concerning results, the author states that "the results of this treatment are so good that I am inclined to doubt the correctness of the diagnosis rather than the effectiveness of the treatment when it fails."

[Comment: Since the usual attack of Ménière's disease is self limited and rarely lasts more than a few hours, it seems doubtful that the improvement observed by the author is the result of his treatment.]

Surgical treatment of Ménière's syndrome by section of the vestibular nerve is recommended by Olivecrona,[76] who has observed unsatisfactory results from medical treatment. Of 21 patients operated on, all but 1 have remained free from attacks of vertigo. By sparing the cochlear nerve the hearing after operation is preserved unchanged and the danger of injuring the facial nerve and the operative risk are rendered slight.

Aubry and Ombredanne [77] employ the intracranial section of the eighth nerve only for marked vertigo which resists other therapeutic attempts. Of 9 patients operated on, 7 experienced relief from their vertigo.

Simonetta [78] reports 2 cases of Ménière's syndrome in which he intended to section the nerve but encountered cystic arachnoiditis of

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75. Leichsenring, E.: Die Behandlung der Ménièreschen Krankheit mit der Pilocarpin Schwitzkur, Med. Klin. **34**:1361 (Oct. 14) 1938.

76. Olivecrona, H.: Ueber Ménière's Krankheit und ihre chirurgische Behandlung, Schweiz. med. Wchnschr. **68**:125-128 (Feb. 5) 1938.

77. Aubry, M., and Ombredanne, M.: Indications et résultats de la chirurgie intracranienne du nerf auditif, Ann. d'oto-laryng., October 1938, pp. 999-1003.

78. Simonetta, B.: Sulle indicazioni della resezione della branca vestibolare dell'acustico nella vertigine de Ménière, Riv. oto-neuro-oftal. **15**:401-407 (Sept.-Oct.) 1938.

the posterior fossa and the patient was cured without section of the nerve. He therefore believes that the nerve should be cut only when no changes of the arachnoid can be demonstrated.

Smith and White [79] report the unusual combination in the same patient of tic douloureux and Ménière's syndrome, the patient having experienced her first and only attack of vertigo two weeks before the fifth and the vestibular portion of the eighth nerve were divided at a single operation with complete and immediate relief of all symptoms.

Putnam [80] describes a new method of surgically treating Ménière's syndrome. A man of 44 with recurrent vertigo was operated on, the intention being to section the eighth nerve. Because of technical difficulties the operation was terminated before the nerve could be exposed. The symptoms continued. At a second operation the dura was elevated over the arcuate eminence through a subtemporal decompression and a burr was used to enter the superior canal. A fine wire was introduced into the canal anteriorly and a light cutting current applied for five seconds. There was no further dizziness, but because the tinnitus persisted the original hole was enlarged, with exposure of the utricle and coagulation for a longer period. Even this failed to destroy all the hearing; so the cochlea was exposed through the same approach and lightly cauterized. The hearing was finally destroyed, but paralysis of the facial nerve lasting six weeks occurred, and the tinnitus persisted. The patient occasionally feels slightly dizzy.

A second patient, a woman of 43 with recurring vertigo following acute otitis media unrelieved by the Furstenberg regimen, was also operated on through subtemporal decompression with opening and coagulation of the superior canal for fifteen seconds. A month later there was a severe attack of vertigo; so the opposite superior canal was operated on in a similar manner. Following this there were severe attacks of vertigo and hearing tests showed partial but not complete deafness in this second, previously well hearing, ear. The tinnitus was unchanged.

The author concludes that in 2 cases "the severe attacks of vertigo have been done away with by a simple, safe operation, which need not affect hearing."

[Comment: The author's enthusiasm for this procedure hardly seems justified by his case reports.]

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79. Smith, H. M., and White, M.: Concurrent Tic Douloureux and Ménière's Disease Treated Surgically, J. A. M. A. **111**:782-783 (Aug. 27) 1938.

80. Putnam, T. J.: Treatment of Recurrent Vertigo (Ménière's Syndrome) by Subtemporal Destruction of the Labyrinth, Arch. Otolaryng. **27**:161-168 (Feb.) 1938.

Peacock [81] injected alcohol through the drum membrane and oval window in 2 patients with aural vertigo, using a needle with a double lumen, so that the labyrinth was rinsed out. After initial symptoms of vertigo, function was completely abolished.

Wright [82] first destroyed the labyrinth by alcohol injected through the oval window in 1935, but permanent paralysis of the facial nerve resulted from the excessive amount of alcohol used. Since then he has reduced the amount of alcohol to 1 minim (0.06 cc.) and in only 1 case has he had temporary paralysis of the facial nerve. This procedure is indicated only for the exceptional patient since removal of foci will cure the majority. When the age of the patient precludes removal of foci or when the vertigo results from suppuration of the middle ear, especially after the radical operation, the procedure is simpler, safer and quicker than section of the eighth nerve.

The author uses an electric otoscope, a special spring-operated syringe with a special trigger release and an angled needle, the point of which is beveled nearly flat. The meatus is cleaned before the operation, but at the operation no preparation is given to the meatus. Absolute alcohol colored with methylthionine chloride (methylene blue) is used for the injection. Light general anesthesia is employed. The oval window lies on a line drawn through the short process of the malleus at an angle of 15 degrees above a line joining the external meatus and the infra-orbital ridge, the latter line being marked by a thin strip of adhesive tape. The needle is inserted at the approximate position of the oval window, and firm pressure is made in one or more positions until the needle is felt to pass through a thin layer of bone and inward 2 mm. This should be practiced first on the cadaver. In the 1 case of temporary paralysis of the facial nerve the needle evidently entered the facial canal instead of the labyrinth, since a sudden contracture of the facial muscles occurred without destruction of the labyrinth. A week later the operation was successfully repeated.

Following the operation there are vomiting and vertigo lasting a few days, the patient being out of bed by the fourth or fifth day. There have been no infections.

Yearsley [83] reports 5 cases in which labyrinthectomy was performed between 1908 and 1933 for vertigo. In all 5 cases otitis media had occurred previously. In each case radical mastoidectomy was followed

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81. Peacock, R.: Alcoholic Labyrinthine Injection Through the Oval Window in the Treatment of Aural Vertigo, Lancet **1**:421-423 (Feb. 19) 1938.

82. Wright, A. J.: Labyrinthine Destruction in the Treatment of Vertigo by the Injection of Alcohol Through the Oval Window, J. Laryng. & Otol. **53**: 594-597 (Sept.) 1938.

83. Yearsley, M.: Operative Treatment of Labyrinthine Vertigo, Lancet **2**: 618-619 (Sept. 10) 1938.

by labyrinthectomy. The author believes that this procedure is simpler and safer than intracranial section of the eighth nerve.

[Comment: In summarizing these interesting contributions on the treatment of Ménière's disease or syndrome—the two terms are applied by different authors to the same condition, and there is some question in my mind which is preferable—there is considerable evidence that focal infection plays a part in at least some cases, that allergy is a factor in others and that there may be a few remaining instances in which surgical treatment is required. Sectioning the vestibular nerve has the advantage of frequently preserving the hearing, but it is a major surgical procedure not without risk. Injection of alcohol through the oval window is a simple procedure, which, however, sacrifices what hearing remains and which may injure the facial nerve. Labyrinthectomy or the subtemporal approach described by Putnam seems to be a more extensive operation than injection through the oval window, with no advantages.]

122 South Michigan Avenue.

News and Comment

INTERNATIONAL COLLEGE OF SURGEONS

The officers of the United States chapter of the International College of Surgeons cordially invite all physicians and surgeons in good standing to their fourth annual assembly, to be held in Venice, Fla., Feb. 11 to 14, 1940. There is no registration fee.

The convention will be under the direction of Dr. Fred H. Albee, of New York, international president-elect, and Dr. Frederick M. Douglass, of Toledo, Ohio, president of the United States chapter.

For general information please address Dr. Fred H. Albee, chairman, 57 West Fifty-Seventh Street, New York. For information about the presentation of scientific papers or exhibits, address Dr. Charles H. Arnold, secretary of the scientific assembly, Terminal Building, Lincoln, Neb.

Abstracts from Current Literature

Ear

EAR COMPLICATIONS OF SCARLET FEVER. A. L. HOYNE and R. SPAETH, J. Pediat. **12**:287 (March) 1938.

The aural complications of scarlet fever are so frequent and so serious that their importance should be appreciated more fully by members of the medical profession. A study was made of the aural complications among 3,564 patients with scarlet fever treated at the Municipal Contagious Disease Hospital in Chicago from July 1, 1934, to June 30, 1935. Tables are presented showing the data on the incidence of otitis media among these patients in comparison with similar data taken from the literature. The incidence of mastoiditis, the number of mastoidectomies done, the mortality, and the complications of mastoid disease in the 3,564 cases also are given in tabular form. Other tables included in the report show the week of onset of purulent otitis media, the incidence according to age and the importance of this complication as a cause of death.

The authors suggest certain therapeutic measures which would in all probability have a distinct influence on the incidence of otitis media. General supportive measures, such as blood transfusions, parenteral administration of fluids, adequate diet and effective sedative therapy, appear to increase the patient's resistance to infections of the middle ear and mastoid. Maintenance of unobstructed nasal passages is essential to the prevention of the mechanical spread of septic material from the nasopharynx through the eustachian tubes to the middle ear. Irrigations of the throat and gargles are to be avoided. In hospital wards patients with uncomplicated scarlet fever should be separated from those with complications, and protection from cross infections is essential. Care must be taken to resort to paracentesis only when necessary, and the operation should be done by an otologist or a competent physician. Irrigations of draining ears have not been done at the Municipal Contagious Disease Hospital during the past eighteen years, but recent reports in the literature on the beneficial action of irrigation with alcohol have been encouraging, and the procedure may be useful. A strict aseptic technic is essential in the care of the draining ear. It is suggested that adenoidectomy be done on patients with otorrhea, in order to decrease the duration of the aural discharge and avoid incriminating invasion of the mastoid cells as a cause of prolonged aural discharge.

RAUH, Cincinnati. [AM. J. DIS. CHILD.]

PARA-AMINOBENZENESULFONAMIDE AND ITS DERIVATIVES IN THE TREATMENT OF BETA HEMOLYTIC STREPTOCOCCUS INFECTIONS OF THE MIDDLE EAR AND MASTOID: REPORT OF SIX CASES IN CHILDREN. C. G. FLAKE and B. W. CAREY JR., New England J. Med. **217**:1033 (Dec. 23) 1937.

Flake and Carey studied the effects of sulfanilamide therapy in 3 patients with meningitis, 1 patient with sterile meningitis, 1 patient with a perisinal abscess and septicemia and 1 patient with postscarlatinal mastoiditis. All the patients recovered. The cases are reported in detail.

In cases 1 and 2 administration of the drug was started subcutaneously and intrathecally. Sulfanilamide was given orally as soon as the condition of the patient permitted. In case 3 subcutaneous or intrathecal treatment with sulfanilamide was not given until twenty-four hours after beta hemolytic streptococci were discovered in the cerebrospinal fluid, but sulfanilamide was given orally in adequate doses during this period.

In case 4 there had been bilateral mastoidectomy and adenoidectomy. Three days after operation meningitic symptoms occurred. The spinal fluid contained

2,800 leukocytes per cubic millimeter (65 per cent polymorphonuclear cells), but smears and cultures were negative. The treatment included administration of sulfanilamide and neoprontosil and blood transfusion.

Case 5 is presented to demonstrate the marked fall in temperature, rapid sterilization of the blood stream and general improvement in a child gravely ill with acute mastoiditis, perisinal abscess and septicemia after simple mastoidectomy with exposure of the lateral sinus and institution of therapy with sulfanilamide and its derivatives.

In case 6 sulfanilamide was given before and after operation.

GENGENBACH, Denver. [AM. J. DIS. CHILD.]

SULPHONAMIDE IN THE TREATMENT OF ACUTE MASTOIDITIS. V. G. HORAN and S. G. FRENCH, Brit. M. J. **2**:942 (Nov. 5) 1938.

These observations were made at the Royal Naval Hospital, Chatham, England, and presumably were made on adults. However, the work probably applies equally to children, although this is not definitely determined. The authors point out that there is a seasonal variation in mastoid complications of middle ear disease. The surgeon of today is apt to adopt an expectant line of treatment rather than the radical one. It is important that administration of sulfanilamide be practiced early rather than late. On account of this practice it may be that an error in estimating results arises in that many who were expected to have mastoid involvement might not have reached that stage in any event. The argument and statement of experience are conservative. In spite of the possible sources of error in the figures published in the report, the conclusions are that the incidence of acute mastoiditis in the Royal Naval Hospital, Chatham, has been notably reduced and that, since the only difference in the treatment of acute disease of the ear has been the introduction of routine administration of sulfanilamide on admission, it is reasonable to suppose that the aforementioned drop in the incidence of mastoiditis is due to the use of the drug. Therefore sulfanilamide and the related drugs have a real place in the treatment of acute suppurative otitis media.

ROYSTER, University, Va. [AM. J. DIS. CHILD.]

PROGRESSIVE FACIAL PALSY PRODUCED BY INTRATEMPORAL EPIDERMOIDS. G. JEFFERSON and A. A. SMALLEY, J. Laryng. & Otol. **53**:417 (July) 1938.

During the last five years Jefferson and Smalley encountered 6 patients in each of whom slowly progressive palsy of the facial nerve was proved to be due to an epidermoid lodged within the temporal bone. No infection was present in any of them, and 3 patients had never had otitis media. All, however, were deaf to a greater or less degree on the side of the palsy, and this fact led to a painstaking investigation of the temporal bone for a possible causative lesion. The paralysis of the facial nerve had in each case taken several months to develop and was in that respect totally unlike Bell's palsy. The syndrome is related to the pure pressure effect of an epidermoid and not to any septic condition such as accompanies the ordinary cholesteatoma.

J. A. M. A.

FLUID BALANCE IN MÉNIÈRE'S DISEASE. T. CAWTHORNE and MARY N. FAWCETT, Lancet **2**:1404 (Dec. 17) 1938.

Cawthorne and Fawcett studied the effect of various intakes of salt and fluid on the symptoms of Ménière's disease in 11 patients. All but 1 were affected to a greater or less degree by variations in the intake of fluid and sodium chloride. Any steps taken to favor the retention of fluid within the body resulted in aggravation of the symptoms, whereas the reverse was true when excretion of fluid was encouraged. In 9 cases this fluctuation of the symptoms was accompanied by variation in the hearing capacity; thus, when the symptoms were more marked the hearing was worse, and vice versa. None of the patients under

review tended to retain fluid unduly, although they were all sensitive to variations in the salt and fluid intake. It seems that imperfect functioning of the pressure-regulating mechanism of the endolymphatic system places the affected labyrinth under the influence of any factors that may affect the secretion or absorption of endolymph on that side. It is not unreasonable to suppose that an increase of the fluid content of the body may favor an increase of the intralabyrinthine pressure. It is recommended that an antiretentional regimen should form part of the investigation of every patient with Ménière's disease. J.A. M. A.

A Review of the Problems of Suppurative Petrositis and of Its Surgical Treatment, Together with an Outline of the Prophylaxis and Treatment of Otitic Meningitis. D. G. Carruthers, M. J. Australia **2**:644 (Oct. 15) 1938.

Carruthers feels that inasmuch as a high percentage of patients who die of otitic meningitis are found at autopsy to have suppurative foci in the petrous pyramid with evidence of direct extension to the meninges, the physician must always consider the possibility of such foci in any case of suppuration of the middle ear in which recovery is not taking place. Petrositis is a comparatively late complication of acute otitis media and may become manifest even after mastoidectomy. The first symptom is pain or aching in the orbit, face, forehead or parietal region on the affected side. Latency is not uncommon when pus first escapes into the meninges. The second symptom is a profuse or moderately profuse purulent discharge, especially one that persists or returns after the mastoid cells have been exenterated. The third symptom, paralysis of the sixth nerve (Gradenigo's syndrome), is not as common as was formerly taught. Low grade pyrexia is common. Transient facial weakness, slight vertigo and nystagmus may be present. Careful and repeated roentgenographic examination may be of great diagnostic help. In interpreting typical signs and symptoms one may have to exclude sphenoid sinusitis, thrombosis of the superior petrosal or cavernous sinus and labyrinthitis. The operative technic developed by Kopetzky and Almour and Lempert is described. In treating the various deep-seated complications of otitis media the surgeon must always watch for signs of early meningitis—any increase in the number of cells in the cerebrospinal fluid and any disturbance in the normal relation of the lactic acid, carbon dioxide, hydrogen ion concentration and chloride content of the blood and cerebrospinal fluid. Once meningitis has occurred, treatment consists in (1) surgical drainage of the feeding focus in the temporal bone, (2) administration of specific antiserum, (3) transfusion of a small amount of whole blood every one or two days and (4) administration of sulfanilamide in large daily doses (from 0.2 to 0.25 Gm.) per kilogram of body weight.

Gonce, Madison, Wis. [Am. J. Dis. Child.]

Septic Meningitis: Report of Case. G. L. M. Scholefield, New Zealand M. J. **37**:35 (Feb.) 1938.

The patient, a man 24 years old, was thrown from a motorcycle on March 29, 1937. He was unconscious for a few minutes and then went home. He remained away from work for one week, during which time frontal headache developed. Physical examination and roentgenograms showed no abnormality, and he signed a release from the hospital after eight days, only to return on April 13 with severe headache, pain in the neck and behind the eyes, photophobia and fever.

Examination showed stiffness of the neck and Kernig's sign on both sides. Lumbar puncture revealed opalescent spinal fluid. The cell count was 1,050 (mostly polymorphonuclears); the globulin test was markedly positive. Organisms were not seen on smear or culture. The right mastoid was opened and appeared normal, but in the roof of the antrum an old clot was found. The dura appeared thick. It was incised, and free pus was obtained from beneath the temporal lobe. This was drained, and thereafter the patient slowly recovered, being given neoprontosil

intramuscularly daily for eleven days and prontosil tablets (form not stated) by mouth. Cultures of the spinal fluid were negative throughout. No mention was made of culture of material from the abscess.

MULHERIN, Augusta, Ga. [AM. J. DIS. CHILD.]

INTRACRANIAL COMPLICATIONS OF EAR DISEASE. J. P. STEWART, Practitioner **141**:603 (Nov.) 1938.

Suppuration occurring in the cleft of the middle ear may give rise to crippling disabilities, such as severe deafness, but it is only when suppuration extends beyond these boundaries that there is danger to life. The intracranial complications which follow acute and chronic suppuration are considered jointly, but there are certain distinctions. In acute infections the complications tend to be diffuse, and in chronic cases more circumscribed, in their initial stages. Thus in chronic cases initial circumscribed meningitis and delayed generalized meningitis and sinus thrombosis occur. Better results from treatment in the chronic cases might therefore be expected, and they do, in fact, occur.

This excellent article should be read in full.

GELSTON, San Francisco. [AM. J. DIS. CHILD.]

THE DEAF CHILD. P. FRANKLIN, Practitioner **141**:615 (Nov.) 1938.

A most interesting part of this article is that relating to mechanical aids to teaching the deaf.

Noises and speech sounds are communicated to the child by two types of electrical amplifiers: (1) electroacoustic; (2) electrovibratory. The electroacoustic apparatus is used to apply stimulation to the natural auditory channel. It is designed to produce sound through a microphone, either from the human voice or from gramophone records. Ear phones are applied to the ears. The electrovibratory apparatus is used to transmit vibrations to the finger tips. These rest on a delicately vibrating metal plate or vibrating cork. By analogy the finger tip serves as the ear and the vibrating plate or cork as the ear phones.

The children are divided into two groups: (1) those who have some remnant of auditory perception; (2) those who have no auditory perception and are therefore trained to receive the impression of sound or human speech through a substitute sense—touch, or the vibrotactile sense. In either group the child is encouraged to watch the teacher of speech form words. His attention is drawn to the appropriate toy, picture or letter spoken. In this way education is assisted as it would be in a normal child.

With reference to the sensory mechanism for feeling vibration, several writers have suggested an analogy between touch and hearing. Touch has been described as primitive hearing which in the course of evolution has been taken over by a specialized organ—the ear—for appreciating vibrations of a limited range—the human range of hearing. Experience with the speech training of deaf-mute children by touch supports the idea of a fundamental relation between the two senses. The use of touch is not new. For centuries deaf-mute children have been placing their fingers on the teacher's larynx to feel the vibrations of vocal sounds.

Emphasis is placed on preschool training, which at first is carried out individually and later in small groups.

GELSTON, San Francisco. [AM. J. DIS. CHILD.]

TUBERCULOUS OTITIS MEDIA. R. B. LUMSDEN, Tr. Med.-Chir. Soc. Edinburgh, 1938, p. 193; in Edinburgh M. J., October 1938.

The clinician presented a boy who had entirely recovered from an extensive radical mastoidectomy for tuberculous disease of the middle ear and gave the opinion that such otitis media is not rare, as he had observed several cases of it.

When a child suffers from an aural discharge that is resistant to treatment, tuberculosis should be thought of, and on repeated examination of the discharge tubercle bacilli may be found. NEFF, Kansas City, Mo. [AM. J. DIS. CHILD.]

TREATMENT OF PURULENT STREPTOCOCCIC MENINGITIS BY SULFANILAMIDE. R. MARTIN and A. DELAUNAY, Ann. méd.-chir. **3**:86 (March) 1938.

Purulent meningitis caused by organisms of the streptococcus group is relatively rare, and the cases constitute only about 6 per cent of all cases of meningitis. In most of the cases the disease is secondary to infectious processes in the ears or nose. The mortality varies from 97 to 100 per cent. Recently, the use of sulfanilamide has been followed by recovery in cases of meningitis of this type. Large doses, up to 0.5 Gm. every two hours, have been given, followed by gradual diminution of the dose to 4 and then to 3 Gm. daily. In infants a dose of 0.001 Gm. per kilogram of body weight has been used. In certain cases intraspinal administration is advisable; the authors advocate the use of from 10 to 20 cc. of an 0.8 per cent solution daily, accompanied by drainage of the cerebrospinal fluid. In all cases in which the meningitis is secondary to a localized infectious process, there should also be surgical intervention.

DE JONG, Ann Arbor, Mich. [ARCH. NEUROL. & PSYCHIAT.]

OTOLOGIC RELATIONS TO DYSENTERY IN INFANCY. B. GALCSIK, Orvosi hetil. **82**:128 (Feb. 5) 1938.

In every case of dysentery an otoscopic examination was made, and if mastoidal empyema was suspected an operation was immediately made, with the infant under local anesthesia. This antrotomy was made even in cases in which the otoscopic examination did not show pathologic changes. In few cases was a healthy antrum found. Up to the years 1930-1932 these operations were rarely performed, and the mortality was 42 to 45 per cent. From 1933 on, when this operation became the rule, the mortality fell to 22 per cent. Galcsik thinks that in this great improvement, apart from the progress in internal treatment, intensive otologic treatment plays a great role. GÖTTCHE, Budapest, Hungary. [AM. J. DIS. CHILD.]

Pharynx

PERIOESOPHAGEAL ABSCESSES: THE IMPORTANCE OF EARLY SURGICAL INTERFERENCE. WESTLEY M. HUNT, Ann. Otol., Rhin. & Laryng. **48**:128 (March) 1938.

The author reports cases and discusses infection of the mediastinum in the cervical region following rupture of the esophagus. He believes that this condition occurred many times prior to the advent of roentgenography and bronchoscopy and not bing recognized was diagnosed as pneumonia or cancer.

Rupture of the esophagus may be caused by: (1) foreign bodies, through immediate perforation, slow erosion or perforation on removal; (2) instrumentation; (3) spontaneous rupture with malignant growths.

Periesophageal abscesses in the cervical region are easily observed, and the surgical approach is easy. First confined to the area between the esophagus and the prevertebral fascia or the esophagus and the trachea, they may descend later.

The diagnosis is made by (1) the suspected or observed perforation; (2) marked collapse of the patient at the time of perforation; (3) pain, tenderness and swelling; (4) inability to swallow; (5) absence of dyspnea unless pneumothorax has occurred; (6) increased leukocytic count—up to 23,000; (7) sudden rise of temperature; (8) roentgen evidence of widening of the prevertebral or post-tracheal space, and (9) emphysema.

Hunt reports Lore's finding that fluid between the esophagus and the trachea tends to point in front of the carotid sheath and anterior to the sternocleidomastoid muscle. Fluid in the prevertebral space tends to point behind the carotid sheath and posterior to the sternocleidomastoid muscle. Lore pointed out also that anterior infections first show tenderness anterior to the sternocleidomastoid muscle and posterior infections posterior to it.

The incision for anterior approach is along the median border of the sternocleidomastoid muscle at the level of the sixth cervical vertebra. The incision is carried downward; the muscles and the carotid sheath are retracted, the middle thyroid vein and the inferior thyroid artery ligated and the thyroid gland retracted. The abscess is opened by blunt dissection, with frequent use of suction. Drains are inserted. The approach on the right side is easier and safer, as the dome of the pleura is lower, the esophagus being in contact with the pleura only in the middle. The thoracic duct is avoided on the right also. Care should be taken to pack off the fascial spaces down to the vertebral column in an effort to prevent a downward spread of infection. If the infection has already passed below, a drainage tube is carefully inserted behind the esophagus to this area.

In the posterior approach the incision is behind the sternocleidomastoid muscle opposite the abscess, and the abscess is approached by blunt dissection behind the carotid sheath. The only structure to avoid here is the cervical ganglion, which is attached to the prevertebral fascia. The brachial plexus is avoided by keeping above the omohyoid muscle.

The following conclusions are reached: 1. A feeding tube should be placed in the esophagus in any case of known or suspected rupture. 2. Daily roentgen examination should be done. 3. Any rise in temperature or white blood cell count, pain or roentgen evidence calls for immediate incision and drainage. 4. Intraesophageal treatment should be used only in most carefully selected cases. 5. It may be better surgical technic to open and pack off any esophageal rupture before abscess has developed.

Twenty cases were analyzed and a number reported. In 16 of the 20 the patient was operated on, with 12 recoveries and 4 deaths. In 4 treatment was through the esophagus. The 4 deaths were in cases in which external drainage had not been established.

M. V. Miller, Philadelphia.

Foreign Body in the Upper Esophagus for Five Months. M. C. Myerson, New York State J. Med. **38**:885 (June 1) 1938.

A white girl aged 6 years complained of severe pain in the region of the upper part of the spine posteriorly. About five months previously she suffered from pain and discomfort in the throat. At that time she visited several dispensaries, without relief. Because of the localized symptoms, a roentgen study was made; a jackstone was found lodged in the upper part of the esophagus. The tracheoesophageal wall was markedly swollen, and it was necessary to insert a flexible tube into the trachea before an esophagoscopic examination could be made. The foreign body was removed successfully, and the child made a good recovery.

Pain and discomfort in the throat of a child should suggest the presence of a foreign body. The same is true of pain in the region of the spine.

Aikman, Rochester, N. Y. [Am. J. Dis. Child.]

Massive Collapse of Lung Following Tonsillectomy. Recovery. R. W. B. Ellis, Proc. Roy. Soc. Med. **31**:772 (May) 1938.

A girl aged 9 years had suffered from asthma between the ages of 3 and 8 years. In December 1936 she had an attack of follicular tonsillitis and subsequently complained several times of sore throat. In April 1937 she was admitted to the hospital for tonsillectomy; on examination mucopus was found under the left middle turbinate and a roentgenogram showed well marked root shadows only. Tonsillectomy was performed and iodized poppyseed oil injected into the maxillary antrums. The operation was apparently uneventful, and bleeding was not excessive.

On the next day, the temperature rose, and the child appeared acutely ill. Movement of the right side of the chest was diminished. There was dulness on percussion. The breath sounds were almost completely absent over the right lung. These signs continued unchanged for three days, when the general condition improved. Roentgen study showed massive collapse of the right lung, with displacement of the heart and mediastinum to the right. Roentgen examination repeated a few days later showed expansion of the upper and the middle lobe and still a few days later reexpansion of the whole of the right lung, with the heart in the normal position. The patient was discharged from the hospital well and free from any abnormal physical signs. WILLIAMSON, New Orleans. [AM. J. DIS. CHILD.]

TUBERCULOSIS OF THE HARD PALATE. M. LANGLOIS, Bull. Soc. de pédiat. de Paris **35**:690 (Dec.) 1937.

A lesion developed on the hard palate of an infant 2 months old. The left cervical glands were enlarged. The patient was admitted to the hospital at the age of 4 months. A biopsy of tissue from the hard palate was made and tuberculosis diagnosed. Later, in pus aspirated from a cervical gland, tubercle bacilli were demonstrated. The tuberculin test was positive. No mention is made of a roentgenogram of the chest. The infant died of tuberculous meningitis following pertussis at the age of 10 months. No contact with any person with open tuberculosis was established. The infant had been fed unpasteurized, unboiled milk from tuberculous cows. BENJAMIN, Montreal, Canada. [AM. J. DIS. CHILD.]

DEMONSTRATION OF PNEUMOCOCCI IN THE THROATS OF NORMAL INFANTS AND OF INFANTS WITH PNEUMONIA OR INFECTION OF THE UPPER RESPIRATORY TRACT. S. NARASAKI, Acta pædiat. japon. **44**:1036 (July) 1938.

Pneumococci were found in the throats of 9.2 per cent (summer) and 2.6 per cent (winter) of all normal infants. In infants suffering from grip the frequency was 16 per cent; in those with bronchitis, 29.2 per cent and in those with pneumonia, 43.3 per cent. These observations prove that with the progress of the disease the frequency of positive cultures increases. The majority of diplococci belonged to type IV. Pneumococci were found in the throats of infants under 1 year of age as frequently as in the throats of older children.

KATO, Chicago. [AM. J. DIS. CHILD.]

Larynx

LARYNGEAL DIPHTHERIA. P. BAYER, South African M. J. **13**:47 (Jan. 28) 1939.

Bayer reports on a study of 1,291 patients with diphtheria admitted to the Fever Hospital, Johannesburg, South Africa, from 1930 to 1937. Of these 242 had laryngeal diphtheria and 124 required tracheotomy. The incidence of diphtheria in Johannesburg is less than 100 cases per hundred thousand of population, whereas that in Great Britain is 140 to 160 per hundred thousand. Diphtheria is almost unknown in children under 6 months of age and uncommon in those under 1 year. Its frequency then increases rapidly, reaching a maximum in children between the ages of 1 and 2 years. Thereafter it declines slowly and in children beyond the age of 8 becomes distinctly rare. The majority of patients have a temperature of 101 to 102 F.; higher temperatures are uncommon. In at least 25 per cent of cases of faucial diphtheria a positive diagnosis can be made immediately from direct examination of a smear. In about 85 per cent of cases of faucial diphtheria culture of swab material is positive after twenty-four hours, and in 95 per cent it is positive after forty-eight hours. In regard to laryngeal diphtheria, however, after tracheotomy cultures from swab material taken from membrane found at operation or from the trachea itself are positive in only 60 per cent of cases. When

the patient was not tracheotomized, swab material gave positive cultures in only 50 per cent of cases. Faucial diphtheria occurs in over half the patients with laryngeal diphtheria and is an additional help in diagnosis. The results of tracheotomy in Johannesburg, with a mortality of 33 per cent, are good in comparison with those of tracheotomy elsewhere but not as good as those of intubation. Whether tracheotomy or intubation is done, the mortality is exceptionally high in children under 2 years of age. Although in very young children intubation gives better results, in older children tracheotomy gives slightly superior results. To a patient with acute streptococcic laryngitis, tracheotomy usually does not bring that intense relief from dyspnea that it gives to a patient with diphtheria. The prognosis in cases of diphtheria must be guarded. Of 41 deaths, 21 were due to spread of the diphtheritic process into the trachea and lungs, 13 to cardiac failure from shock and toxemia and 5 to bronchopneumonia. The complication of bronchopneumonia is by no means always fatal; of 13 patients only 5 died. The first essential in treatment is to regard laryngeal obstruction in a child as diphtheritic until it is proved otherwise. With faucial involvement a large dose of antitoxin, as much as 50,000 units, should be given. Experience is the best aid in deciding whether to perform a tracheotomy and when. Tracheotomy performed after intubation carries a frightful mortality. The author prefers low tracheotomy.

GONCE, Madison, Wis. [AM. J. DIS. CHILD.]

CONGENITAL LARYNGEAL STRIDOR. D. N. NICHOLSON, Tr. Med.-Chir. Soc. Edinburgh, 1938, p. 191; in Edinburgh M. J., October 1938.

Nicholson presented an infant of 6 months with congenital laryngeal stridor, a usually harmless type of noisy inspiration, which alarms the parent. The stridor is at its maximum at about the sixth month of life, having become louder as the child got stronger. It slowly wanes, to disappear by the end of the second year. The child is not disturbed by the stridulous condition. Congenital webbing and papilloma must be ruled out in the diagnosis.

NEFF, Kansas City, Mo. [AM. J. DIS. CHILD.]

CLINICAL, ANATOMOPATHOLOGIC AND BACTERIOLOGIC STUDY OF TWO CASES OF ACUTE STENOSING LARYNGOTRACHEOBRONCHITIS, CHEVALIER JACKSON TYPE. P. ROHMER and C. OBERLING, Bull. Soc. de pédiat. de Paris **36**:242 (April) 1938.

Two girls, 6 years and 11 months old, respectively, died of acute suffocative tracheobronchitis. Necropsy confirmed the diagnosis. Abundant mucofibrinopurulent exudate completely obstructed the air passages in many places. In the older child the trachea and bronchi showed the most involvement, while in the younger one the small and medium-sized bronchioles were affected, and there were a few scattered areas of bronchopneumonia. In the latter case cultures made from the interior of the lung gave growths of Staphylococcus albus, Streptococcus haemolyticus, Friedländer's bacillus and a gram-negative bacillus of the colon group which did not ferment maltose. Experiments with the strain of Friedländer's bacillus which was isolated showed it to be virulent for mice.

BENJAMIN, Montreal, Canada. [AM. J. DIS. CHILD.]

Nose

NASAL SEPTUM SURGERY IN CHILDREN. M. H. COTTLE, Illinois M. J. **75**:161 (Feb.) 1939.

After a discussion of the histologic aspects, causes, symptoms and treatment of deviations of the nasal septum, Cottle concludes that frequently nasal obstruction

and chronic discharge are the result of septal deviation, that often nonsurgical treatment is needed and is adequate, that occasionally surgical measures are indicated and that the type of operation which is appropriate to an adult is unnecessarily radical for a child in all but the rare instances of extreme deformity.

BARBOUR, Peoria, Ill. [AM. J. DIS. CHILD.]

SINUSITIS IN CHILDREN. E. C. MITCHELL, J. A. M. A. **112**:207 (Jan. 21) 1939.

Sinusitis in children presents a favorable outlook for recovery and development of the sinuses if the condition is cared for early. Early recognition of the etiologic nature is important, especially if this is allergic. The treatment varies with the stage of the disease; it is outlined for the acute, subacute and chronic stages. The condition should be followed by roentgen examinations and observations for foci of infection.

HEERSMA, Kalamazoo, Mich. [AM. J. DIS. CHILD.]

PARANASAL SINUSES IN CHILDREN. A. BOWEN-DAVIES, Proc. Roy. Soc. Med. **31**:1411 (Oct.) 1938.

Bowen-Davies made a routine examination of 55 children between the ages of 5 and 14 years. A roentgenogram was taken; the nose was cocainized and swabbed, and both antrums were punctured. The washings were cultured and the antrums filled with iodized poppyseed oil, 40 per cent. Further roentgenograms were taken at once and at intervals of a week or so until the antrums were clear of iodized oil. The volume of iodized oil and the time it took to disappear were recorded. Organisms were found in the antrums of 23 patients and mucopus in the nose of 19. Cultures of the mucopus from 6 of these proved to be sterile. The organisms found in the antrums, both those in which the mucopus was infected and those in which it was sterile, were similar to those present in the nose. Five tables are given, listing the organisms found in the various cases, together with a series of roentgenograms showing the progress of the evacuation of the iodized oil. In all cases improvement followed the injection of iodized oil, not only in the clinical condition of the patient but also in the roentgenographic appearance of the antrums. Local treatment is of established value, and the removal of tonsils and adenoids may be advisable. An abundance of fresh air and an adequate diet, with a plentiful supply of vitamins may do a great deal to prevent sinusitis in children.

WILLIAMSON, New Orleans. [AM. J. DIS. CHILD.]

A CASE OF ACUTE FRONTAL SINUSITIS TERMINATING IN DEATH: STATISTICAL STUDIES. SOJI SHIMIZU, Oto-rhino-laryng. **12**:18 (Jan.) 1939.

In a 19 year old barber purulent sinusitis on the right followed a cold. A week later edema developed over the right side of the forehead and the right upper eyelid; the patient had a headache and a septic temperature. In ten days he was hospitalized with a diagnosis of frontal sinusitis on the right. With the patient under local anesthesia a classic incision was made for opening of the frontal sinus. About 30 cc. of thick pus was evacuated from the subcutaneous tissue, but the abscess did not have an apparent connection with the frontal sinus. Closer examination revealed that the periosteum was lacking over the right orbital rim. The anterior plate of the frontal bone did not show any marked change other than acute congestion and a suggestion of slight softening. The mucous membrane of the sinus was hyperemic, particularly over the area where the periosteum was lacking. The cavity contained a large amount of thick, brownish pus. The frontal nasal duct was patent. The operation ended with exenteration of the anterior ethmoid cells. The exciting organisms were Streptococcus haemolyticus and Diplococcus pneumoniae. The septic temperature continued after the operation, and the patient died on the ninth postoperative day, of bronchopneumonia.

The writer reviewed 47 cases of frontal sinusitis, acute and chronic, in the Japanese literature. The incidence among males was 69 per cent, the condition being more common among the young and middle aged and occurring more frequently on the left than on the right. In most cases there was acute exacerbation of a chronic condition. The acute condition was complicated by subcutaneous abscess of the forehead and orbital abscess; the chronic, by subcutaneous abscess and optic neuritis. Bony fistula of the frontal sinus occurred in the anterior or posterior table in 3 cases, in the superior wall of the orbit in 2 and in the ethmoid plate in 6. The mortality was 8.5 per cent.

HARA, Los Angeles.

Miscellaneous

INTRAVENOUS INJECTION OF HYPOTONIC SALT SOLUTION CONTAINING SULFANILAMIDE FOR STREPTOCOCCIC MENINGITIS. G. M. RETAN, Am. J. Dis. Child. **56**:483 (Sept.) 1938.

In experiments on monkeys, planned to determine whether giving sulfanilamide intravenously has the advantage of increasing the concentration of the drug in the cerebrospinal fluid, Retan observed that when physiologic solution of sodium chloride containing sulfanilamide is given to monkeys intravenously the concentrations of sulfanilamide are greater in the blood than in the cerebrospinal fluid. When a hypotonic solution of sodium chloride containing sulfanilamide is injected intravenously, the concentrations during the first injection are the same as with the physiologic solution. However, if the injection of a hypotonic solution is repeated on the second day there occurs a shift to a higher percentage of sulfanilamide in the cerebrospinal fluid than is contained in the blood. A similar shift to a higher concentration in the cerebrospinal fluid than in the blood can be produced by giving the drug by mouth in solution or in capsules prior to the intravenous injection of a hypotonic solution of sodium chloride. The shift depends on the presence of sulfanilamide in the tissues of the body and the intravenous introduction of a hypotonic solution of sodium chloride containing sulfanilamide. The author is not in a position to know what result he would have obtained in his case of hemolytic streptococcic meningitis had he given sulfanilamide by mouth and by subcutaneous injection without using injections of hypotonic solution of sodium chloride containing sulfanilamide. There was prompt subjective and objective improvement within an hour after the beginning of each intravenous injection. The doses of sulfanilamide used in the intravenous solution (from 6 to 10 Gm. in twenty-four hours) were unnecessarily large. An alkaline powder, of calcium carbonate and sodium bicarbonate, was given at intervals of four hours. The rate of injection most successfully used is 10 cc. of solution each hour per pound of body weight. A 0.375 per cent solution has been found to be most effective. Treatment for five hours with several hours of rest between treatments is advised. Cerebrospinal fluid is released from the lumbar puncture needle during the intravenous injection for the purpose of relieving increased intracranial pressure. If the fluid is under considerable pressure and is spurting from the needle, it is wise to drain from 5 to 10 cc. during half an hour. If the fluid is dripping rapidly, less than this amount should be drained. If the fluid emerges from the needle by a slow drop the stylet should be replaced at once without draining any fluid.

J. A. M. A.

DIPHTHERITIC INVOLVEMENT OF THE LIPS, WITH ABSENCE OF SIGNS IN THE NOSE AND THROAT. H. J. LAVENDER and J. B. SQUIRES, J. A. M. A. **111**:915 (Sept. 3) 1938.

A 10 year old Negro girl had a granular membranous lesion on the lower lip, which had developed twelve days after hospitalization for a severe burn. Cultures of material from the throat were negative until eleven days later. The history of

the case is given, the only one ever to have been reported in which diphtheria of the lip (and of the cheek, gums and alveoli as well) was present without the organisms being found in the throat.

HEERSMA, Kalamazoo, Mich. [AM. J. DIS. CHILD.]

PERSISTENT ANOSMIA FOLLOWING ZINC SULFATE NASAL SPRAYING. F. F. TISDALL, A. BROWN and R. D. DEFRIES, J. Pediat. **13**:60 (July) 1938.

During the outbreak of poliomyelitis in Toronto in 1937 the zinc sulfate nasal spray for prophylaxis was used on a group of 4,713 children from 3 to 10 years of age. These children received two sprayings ten to twelve days apart, and 520 additional children of the same range of age received one spraying. The treatments were conducted in special clinics by otolaryngologists. Not more than 25 per cent of the children experienced temporary anosmia. Few, if any, reported subsequently to the various hospital clinics complaining of persistent anosmia.

Thirty-two otolaryngologists reported on the children treated by them in private practice. At the end of two months 36 children complained of anosmia, with or without disturbance of taste, and at the end of six months 52 reported anosmia. The complaint of persistent anosmia occurred more frequently among older children; only 5 under 10 years of age were reported as having anosmia at the end of two months and only 1 under that age at the end of six months. Persisting disturbance of taste and smell was noted in 11 patients who received only one spraying and in 39 to whom two sprayings were given. Of the 52 persons who had disturbances of smell and taste, some suffered marked loss of smell and in others smell was returning. Some complained of unpleasant odors and others of a distorted sense of smell. The persistence of a disturbance in smell and taste after a period of six months is difficult to explain on the basis of persistence of inflammation of the olfactory mucous membrane causing mechanical blocking of the olfactory area. The effect of a 1 per cent solution of zinc sulfate on the mucous membrane of the human being should be carefully studied to determine whether persisting disturbance of smell and taste may be due to injury of the olfactory area.

RAUH, Cincinnati. [AM. J. DIS. CHILD.]

SULFANILAMIDE IN THE TREATMENT OF ACUTE INFECTIONS OF THE CENTRAL NERVOUS SYSTEM. J. B. NEAL, E. APPELBAUM and H. W. JACKSON, M. Clin. North America **22**:1419 (Sept.) 1938.

The value of sulfanilamide in the treatment of virus diseases is problematic. Also, its value in the treatment of meningitis due to the staphylococcus, the influenza bacillus or the nonhemolytic streptococcus is doubtful. The pneumococcus likewise seems resistant to sulfanilamide since, of 25 persons with pneumococcic meningitis in whom it was employed, only 4 recovered. While the experience of the authors with sulfanilamide in the treatment of meningococcic meningitis has been limited and the fatality high, they believe that it has a favorable effect on the disease and may well replace intravenous use of serum in cases of meningococcemia with or without meningitis. Most encouraging results were obtained in the treatment of meningitis due to infection with the hemolytic streptococcus. Earlier, during more than twenty-six years, the authors observed 274 cases of the various forms of streptococcic meningitis; recovery occurred in only 15 cases, 9 of which were definitely cases of infection with the hemolytic type of streptococcus. Since 1937 they have treated 19 patients with sulfanilamide, with 14 recoveries and 5 deaths.

The preparations used were neoprontosil given intramuscularly, sulfanilamide given orally and at times an 0.8 per cent solution of sulfanilamide crystals given intraspinally. As a rule, 5 cc. or less of neoprontosil was given to younger children and 10 cc. every four hours to older children and adults. In addition, from 5 to 15 grains (0.3 to 1.0 Gm.) of sulfanilamide was given by mouth every six hours. This combination seems to be more effective than either preparation alone.

The advantage of giving the drug intraspinally is open to question. The large doses advocated by some do not seem to be necessary. The authors emphasize that in the treatment of meningitis caused by the hemolytic streptococcus it is of great importance to remove the primary foci of infection.

EUGENE H. SMITH, Ogden, Utah. [AM. J. DIS. CHILD.]

TREATMENT OF PNEUMOCOCCIC MENINGITIS. M. FINLAND, J. W. BROWN and A. E. RAUH, New England J. Med. **218**:1033 (June 23) 1938.

Of 99 patients with pneumococcic meningitis, none recovered except 6 of the 10 who were treated with sulfanilamide alone or with serum. The procedure adopted was as follows: 1. Complete and frequent drainage of the spinal fluid was carried out. 2. Continuous large doses of sulfanilamide were given by mouth or by subcutaneous injection, if necessary, immediately. The optimal dose has not been determined. Sodium bicarbonate was given with each dose. 3. The pneumococcus was identified as rapidly as possible, and sufficient specific antipneumococcus serum was given intravenously to establish a balance of antibody in the circulating blood. 4. Moderate intake of fluid was maintained. 5. About two hours after a reasonable dose was given, blood was withdrawn, and the serum was separated. 6. At the time of the next lumbar puncture this serum was given intraspinally (from 5 to 10 cc.). 7. Lumbar punctures were repeated until the fluid was normal. The frequency was determined by the initial pressure of the fluid and its cellular and protein contents. 8. After the first week of sulfanilamide therapy, if anemia developed transfusions were given and were repeated as necessary until the use of the drug was discontinued (after from seven to fourteen days, at which time the spinal fluid was sterile). Such procedures serve to insure a balance of antibody in the blood stream and to control the bacteremia. They should, in most instances in which the sulfanilamide effectively reduces the infection, provide an adequate amount of antibody and complement in optimal proportions and in a medium which is likely to give the least local or general reaction and the greatest antibacterial effect.

J. A. M. A.

VERTIGO: ITS NEUROLOGICAL, OTOLOGICAL, CIRCULATORY, AND SURGICAL ASPECTS. W. RUSSELL BRAIN, Brit. M. J. **2**:605 (Sept. 17) 1938.

Brain points out that vertigo may arise as a result of disturbances of function at various levels of the nervous system. His classification includes: (1) psychogenic vertigo, (2) vertigo due to cortical disturbances, (3) vertigo of ocular origin, (4) vertigo of cerebellar origin, (5) vertigo due to lesions of the brain stem, (6) vertigo due to lesions of the eighth nerve and (7) aural vertigo. An analysis of 41 cases indicates that the average age of onset is 49 years. Brain believes that focal sepsis is the most important etiologic factor. Most patients respond well to some form of medical treatment, but surgical division of the vestibular fibers of the eighth nerve occasionally is necessary.

ECHOLS, New Orleans. [ARCH. NEUROL. & PSYCHIAT.]

A SIMPLE METHOD OF BRONCHOGRAPHY FOR CHILDREN. N. M. JACOBY and G. KEATS, Lancet **2**:191 (July 23) 1938.

If iodized poppyseed oil 40 per cent is introduced into the pharynx of a child under general anesthesia it must enter the trachea. In 34 cases this principle has been applied to bronchographic study, the children being anesthetized with avertin with amylene hydrate and ether and the oil injected into the pharynx through the mouth with a syringe and cannula. The method has been found to be satisfactory and free from danger.

The child is given a course of postural drainage for several days before bronchographic study is attempted. The only thing to be guarded against at the time of bronchographic examination is the collection of iodized oil between the

cheek and gums, but this will not happen if the point of the cannula is kept close to the posterior pharyngeal wall. The amount of iodized oil to be used varies with the size of the child, but the authors have never used less than 3 or more than 8 cc. for each side.

The child is kept in the semisitting position for about half a minute after the injection; he is then laid down on his back, his side or his face, depending on which lobe it is desired to fill. In many cases he is laid consecutively in all positions. An interval of two to three minutes must be allowed to elapse between injection of the iodized oil and the taking of roentgenograms.

The induction of general anesthesia in children who already have pulmonary lesions may appear to be contraindicated, but the authors believe that this objection is more theoretic than actual. On rare occasions general anesthesia is contraindicated.

LANGMANN, New York. [AM. J. DIS. CHILD.]

PURULENT MENINGITIS CURED BY SULFANILAMIDE: DISCUSSION ON THE NATURE AND POSSIBLE RELAPSE OF STREPTOCOCCIC MENINGITIS. R. MARTIN and A. DELAUNAY, Bull. Soc. de pédiat. de Paris **36**:107 (Feb.) 1938.

A boy 8½ years old had streptococcic meningitis, from which he recovered after oral administration of sulfanilamide. Seven months after the onset of this disease purulent meningitis developed a second time, associated with rhinopharyngitis. No bacteria were demonstrated in smears or cultures of the spinal fluid during this recurrence of meningitis, but it is believed that the infection which was shown to be responsible the first time remained latent and later flared up, causing a recurrence of meningitis. The patient was again given sulfanilamide by mouth and again recovered.

BENJAMIN, Montreal, Canada. [AM. J. DIS. CHILD.]

OCULAR SYMPTOMS IN EPIPHARYNGEAL TUMORS: REPORT OF CASES. E. CUSTODIS, Klin. Monatsbl. f. Augenh. **101**:49 (July) 1938.

In an introduction Custodis explains the importance of tumors of the epipharynx by the proximity of this organ to the base of the skull and the sphenoid bone. The observation of 4 cases prompts him to draw the following conclusions: Benign as well as malignant tumors of the epipharynx may produce disturbances of the sensory, motor and sympathetic nerve fibers connected with the eye. While the malignant neoplasms of the epipharynx may be characterized by a certain complex of symptoms, the benign tumors manifest themselves in various clinical forms. Unexplained disturbances of the eyes and of the nerves supplying the orbit should prompt an examination of the epipharynx by the rhinologist. Roentgenographic examination may facilitate the diagnosis. The characteristic changes produced by a tumor of the epipharynx can be recognized in the axial view of the base of the skull and on anteroposterior exposure of the skull.

K. L. STOLL. [ARCH. OPHTH.]

Society Transactions

MONTREAL MEDICO-CHIRURGICAL SOCIETY, SECTION OF OTOLARYNGOLOGY

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April 14, 1939

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Nasal Sinusitis and Asthma: A Thesis. Presented by Dr. Francis M. Rackemann,* and Dr. Francis L. Weille,† Boston.

In a recent paper on "Intrinsic Asthma," one of us (Rackemann, F. M.: Intrinsic Asthma, *J. Allergy,* to be published) presented the reasons for thinking that asthma might have a cause different from allergy. Certainly there are cases in which it is quite impossible to show a cause which is allergic in the ordinary sense. Whereas allergy is often an adequate explanation of asthma, there is reason to think that it is only one of several causes which can precipitate the asthmatic syndrome.

The relation of the nose and throat to asthma is important for two principal reasons: 1. Lesions of the sinuses and polypi in the nose are so common as almost to be expected in cases in which asthma has become severe. 2. In the presence of a lesion, it is always tempting to both physician and surgeon to advise radical operative treatment, in spite of abundant evidence that results are not often good. Let us review the knowledge of this subject briefly.

There are three ways in which disease of the paranasal sinuses can theoretically cause asthma. First, chronic inflammation can give rise to an exudate which drips down the back of the throat to cause a chronic irritation of the trachea and larger bronchi. Second, inflammation of the sinuses can cause irritation of the nerves in the nose, from which impulses can pass by reflex pathways through the sphenopalatine ganglion to produce overactivity of the parasympathetic (vagus) system and so cause bronchospasm by increased muscular and glandular excitability. Third, chronic sinal infection can act as a focus of infection from which bacteria and products of their metabolism may enter the lymphatics and the blood stream to set up sensitization and subsequent allergic responses in the bronchial structures.

In case any one of these theories was good, one would expect that treatment of the sinal infection would bring relief to the asthma, and one must admit that in some cases surgical intervention does good. In any large series of cases, however, particularly when the patients are followed for as long as two years after the operation, the end results are often disappointing. The reasons for failure are numerous. First, whether the operation is conservative or radical, it is next to impossible to remove all of the septic process. The focus persists. Moreover, even if removed at first, it may recur. Second, radical treatment may change the structure of the nose so much as to impair its normal function seriously. It is conceivable that scar tissue can in itself lead to irritation of nerve endings and so promote asthma by reflex mechanisms. Third, postoperative care is almost as important as the operation, for without it healing is slow, discharge from the sinuses continues and the postnasal drip continues to cause trouble. Finally, the failure of sinal operation may well depend on the thesis, first mentioned in 1929

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by Rackemann and Tobey, that disease of the sinuses in asthma is a part of the picture and not a cause of it. The object of this paper is to present further evidence to support this thesis.

Incidence.—In 1929 F. M. Rackemann and H. G. Tobey (Studies in Asthma: IV. The Nose and Throat in Asthma, *Arch. Otolaryng.* **9**:612 [June] 1929) reviewed 1,074 cases of asthma in which the patients had been followed carefully for two years and found evidence of sinusitis in 271 cases, or 25 per cent. In 1932 R. A. Cooke (Infective Asthma: Indications of Its Allergic Nature, *Am. J. M. Sc.* **183**:309, 1932) commented on the variation of sinal disease with the age of the patient. Sinal disease was found in 38 per cent of young persons, whose asthma began between the ages of 10 years and 30, but when the onset was later, between 30 and 50, the incidence was raised to 65 per cent. When it was after 50, sinal disease was found in 85 per cent. W. T. Vaughan (Some Rhinologic Aspects of Allergy, *J. Allergy* **4**:127, 1933) studied 132 cases which included instances of asthma of all kinds and affecting patients of all ages. Thirty per cent of the patients had had previous operations without benefit, and at the time of study 94, or 71 per cent, presented evidence of nasal disease. S. F. Kelly's (Incidence of Sinusitis and Nasal Polypi in Bronchial Asthma, *Laryngoscope* **46**:692, 1936) figures are a little higher, for of his 100 patients 89 had hyperplastic involvement of the sinuses and only 11 were free of trouble. In a clinic specially planned for nasal studies in asthma, F. L. Weille (Studies in Asthma: XIX. The Nose and Throat in Five Hundred Cases of Asthma, *New England J. Med.* **215**:235, 1936) in 1936 found that 70 per cent of 500 patients with asthma of all ages had sinusitis.

S. S. Bullen (Incidence of Asthma in Four Hundred Cases of Chronic Sinusitis, *J. Allergy* **4**:402, 1933) approached the problem from the opposite direction. He tried to find how many patients with sinal disease had asthma in addition. He studied 400 patients with definite sinusitis and found that only one quarter had evidence of any pulmonary disease and that only an eighth (12.2 per cent of the whole) had asthma. The point was made that in only 8 per cent did the asthma begin at the same time as the sinusitis. One can say, therefore, that in asthma sinal disease is common but that in sinal disease asthma is only one of numerous complications. To have asthma without sinual disease is unusual, but to have sinal disease without asthma is common.

Pathologic Picture.—The pathologic picture of nasal sinusitis has been described, and the literature has been reviewed in great detail, in the book by F. K. Hansel (Allergy of the Nose and Paranasal Sinuses, St. Louis, C. V. Mosby Company, 1936) and more recently in an excellent article by H. Semenov (The Surgical Pathology of Nasal Sinusitis, *J. A. M. A.* **111**:2189 [Dec. 10] 1938).

There are two principal varieties of sinal disease. One depends obviously on an infection of the mucous membrane and is associated with pus. The cells of the exudate are mostly neutrophilic polymorphonuclears, and bacteria are easily cultured. This is the common form which, according to H. Semenov (The Surgical Pathology of Nasal Sinusitis, *J. A. M. A.* **111**:2189 [Dec. 10] 1938), occurs in 50 per cent of cases. Nonpurulent sinusitis, sometimes called hyperplastic or polypoid, may result from a purulent process, or it may depend on some other mechanism, not clearly understood but recognized as concerned with allergy. In all cases, trouble begins with edema of the mucous membrane. This may depend on impairment of circulation, perhaps small thromboses in the capillary walls; it may depend on abnormal capillary leakage, or it may depend on obstruction to the normal lymphatic drainage. Once started, edema results in degeneration of the epithelium and of the basement membrane under it. In purulent sinusitis the edema becomes infiltrated with polymorphonuclear leukocytes, whereas in hyperplastic or allergic sinusitis eosinophils and eosinophilic plasma cells are widely distributed and become a characteristic part of the picture. In cases of acute involvement the whole process may subside without harm, but in other cases connective tissue infiltrates the swollen areas; cysts of various kinds are formed, and, more characteristic, a state of chronic hyperplasia, referred to as thickened mem-

brane in the roentgenologic report, takes its place. As the surface of the membrane forms crypts and pockets, it becomes irregular and polypi form as a part of the degenerative and later of the reparative process. Thickened membrane may harbor pockets of pus within itself, as shown originally by F. L. Weille (Asthma: XI. The Pathology of Allergic Tissue as Seen in the Nose and in the Accessory Sinuses, *Arch. Otolaryyng.* **12**:785 [Dec.] 1930) and later by R. C. Grove and R. A. Cooke (Etiology and Nature of Chronic Hyperplastic Sinusitis, *Arch. Otolaryng.* **18**:622 [Nov.] 1933). Weille found that these pockets occurred in about 10 per cent of cases. Cultures of material from the sinuses were studied by Grove and Cooke. They cultured the washings of the sinuses and compared them with other cultures made from the lining membranes themselves. In 60 per cent of the cases the same organism was found in both specimens, but in the others the organisms were different. In about a third of the cases cultures from the membranes were sterile. They stated the belief that polypi depended on the infectious element in the process. Semenov (The Surgical Pathology of Nasal Sinusitis, *J. A. M. A.* **111**:2189 [Dec. 10] 1938) stated the belief that in two thirds of the cases hyperplastic sinusitis without evidence of gross infection is due to allergy. It is easy to understand that anatomic obstructions like deviations of the septum interfere with proper aeration and drainage and so aggravate all the processes, including edema, inflammation and degeneration.

Nasal polypi are quite as important as the sinal infection and more characteristic of asthma. T. E. Walsh and J. R. Lindsey (Cytology of Nasal Polypi, *Arch. Otolaryng.* **20**:649 [Nov.] 1934) described two types. In those few which result from obvious infections, the cells in the polypi are neutrophils, and removal does good. In the more common variety associated with allergy, the tissue is full of eosinophils, and removal helps for a time only.

Polypi have been studied carefully by R. A. Kern and H. P. Schenck (Importance of Allergy in Etiology and Treatment of Nasal Mucous Polyps, *J. A. M. A.* **103**:1293 [Oct. 27] 1934). In an important paper, they pointed to the discrepancy between their common occurrence in conditions of purely extrinsic origin and their rarity in the ordinary infectious processes. In asthma the incidence of polypi is high (30.5 per cent). In hay fever and chronic vasomotor rhinitis it is lower (13 and 14 per cent, respectively), and in nonallergic diseases, especially in ordinary sinal infections, the finding of polypi is unusual. These figures, they said, lead to the conclusion that polypi are of allergic origin.

The results of operation on sinal lesions throw a little light on the nature of these processes. In 1933, F. L. Weille (Studies in Asthma: XVIII. The Surgical Treatment of Chronic Sinusitis in Asthma, *J. A. M. A.* **100**:241 [Jan. 28] 1933) made a careful study of 40 patients with asthma on whom he had operated for chronic sinal involvement. Five of them were cured of their asthma, and 9 were markedly improved; 6 were moderately improved, but the other 20 were not affected. Moreover, even of the patients for whom good results were obtained, when they were followed a little longer time, several had relapsed—the asthma had recurred. On the other hand, the sinal disease itself was often benefited, and after operation the lesions became easier to control. Paradoxic were two interesting observations: Of 3 patients having severe asthma and bilateral pansinusitis, operation on one side only resulted in marked improvement of the asthma in 2 and moderate improvement in the third. Second, a number of patients studied later seemed to have an excellent condition in the nose, with all evidence of disease in the sinuses cleared away, but in spite of this their asthma continued quite as before.

In 1935 R. A. Cooke and R. C. Grove (Relation of Asthma to Sinusitis with Special Reference to the Results from Surgical Treatment, *Arch. Int. Med.* **56**: 779 [Oct.] 1935) reported on 120 cases, in 70 per cent of which good results were obtained surgically. They emphasized the importance of complete operation, which produced a good result in 86 per cent, as contrasted with a good result in only 39 per cent of the cases in which surgical treatment was incomplete. Weille's later (1936) figures (Weille, F. L.: Studies in Asthma: XIX. The Nose and

Throat in Five Hundred Cases of Asthma, *New England J. Med.* **215**:235, 1936) are much less favorable. His series comprises 485 patients. Of these, 290 (60 per cent) had no operation. A few had tonsillectomies, and others had extraction of bad teeth. What interests us is the 100 patients for whom operation was done on one or more of the sinuses. Seventeen of these 100 had extrinsic asthma, and so far as the asthma was concerned operation produced a "cure" in 3 cases and improvement in 8 others. Eighty-three of the patients had intrinsic asthma, and in these the end results for the asthma included 7 "cures" and 47 patients improved. In 29 patients, however, the asthma was unchanged, and in some cases it was worse after operation than before. Some of the paradoxic results mentioned by Weille in an earlier paper deserve further consideration and will be mentioned presently.

More recently N. Fox and J. W. Harned (Treatment of Asthmatic Patients in Otolaryngologic Practice, *Arch. Otolaryng.* **25**:393 [April] 1937) have rendered an interesting analysis of patients, with rather startling results. In a group of 150 patients whose sinuses required operation, improvement occurred in from 32 to 60 per cent, according to the operation performed, intranasal exenteration of the ethmoid sinuses being much less effective than total exenteration of all the sinuses at the same time. However, in a similar group of 150 patients who were treated by "medical means," many methods being used, the results were poor in every case. The total duration of the follow-up was not stated.

Thesis.—The thesis proposed here is that asthma, nasal sinusitis with nasal polypi, blood eosinophilia and the peculiar debility called "allergic toxemia" are all parts of a syndrome which depends fundamentally on some "x factor" as yet unknown. Allergy, which has been studied so extensively, is only one of many exciting factors which may precipitate symptoms in the presence of the "x factor." Other excitants are much more simple. They include head colds, fatigue, emotional strain and even changes in weather and temperature.

This conception has developed from a study of the cases of intrinsic asthma (Rackemann, F. M.: Intrinsic Asthma, *J. Allergy,* to be published) in which, by the circumstantial evidence provided by the history and the daily progress of the patient, the influence of foreign substances, both in dusts and in foods, can be excluded. The patients were divided into groups according to their clinical histories. In a few, the asthma had changed its character from isolated attacks of extrinsic origin, each dependent on exposure to a particular foreign substance, to a chronic persistent disease. In others, the asthma had from the first borne a close relation to colds in the head, which occurred in some only two or three times a year but in others every few weeks, especially in winter. In a third group, which included more men than women, the asthma had begun suddenly, usually after the age of 45.

Finally, a group of particular interest was that in which asthma, usually of a severe type, developed rather suddenly in persons, mostly women, who had suffered for some years with chronic persistent vasomotor rhinitis. The table in the paper is summarized here. The figures in this table deserve comment. First, almost half of the total number had a positive family history of allergy, and something over a quarter had positive cutaneous reactions to one substance or another. How can these two items be correlated with the diagnosis of intrinsic asthma? The factor of inheritance is not incompatible with the thesis, for our hypothetic "x factor" may well be something which is inherited. The reference of R. A. Kern and H. P. Schenck (Importance of Allergy in Etiology and Treatment of Nasal Mucous Polyps, *J. A. M. A.* **100**: 241 [Jan. 28] 1933) to polypi in allergy applies equally well to polypi in asthma. The many positive cutaneous tests are excluded for the reason that the particular substances reacting could not be shown to be concerned with the patients' symptoms. We find that many positive cutaneous reactions occur even in normal persons without symptoms to go with them.

The right half of the table is more interesting. Thirty per cent of the patients had had previous operations on the nose and throat, obviously without benefit.

Furthermore, 45 per cent, or almost half, showed lesions in the nose and sinuses at the time of examination. These figures have varied somewhat in the different groups.

About half of those included under "Asthma after colds" had had only two or three colds a year; the term "asthmatic bronchitis" was applicable.. The disease was not severe, and, as expected, only about one third had had or showed any trouble with their sinuses. In none of this subgroup was true pansinusitis observed. Two of the patients had had polypi removed in the past, and 1 had polypi at the time of examination. About one fifth of the patients had had previous operations on the nose and throat—submucous resections, polypectomy, and excision of an antral cyst—but nothing more radical than these procedures, and it is important to note that in none of the cases did these previous operations afford any benefit so far as preventing further attacks or modifying the course was concerned. In the other half of the group, in which colds were more frequent and the asthma more severe, lesions of the nose and sinuses occurred in 39 of the 69 patients. About two thirds of the lesions consisted of pansinusitis, with the other third being merely thickened membrane in the sinuses. Previous operations had been done on about half of the patients who had lesions, most of them being simple polypectomy, but 4 radical sinal exenterations had been done, without benefit.

Intrinsic Asthma

	Number of Cases	Number of Males	Average Age at Onset	Family History of Allergy	Cutaneous Tests Showing Allergy	Previous Operation on the Nose and Sinuses	Present Lesions in the Nose and Sinuses	Death from Asthma
Extrinsic asthma changing to intrinsic	16	7	50	10	10	4	4	3
Asthma after colds	135	52	31	55	26	32	50	3
Sudden onset	83	45	50	27	22	16	40	4
Vasomotor rhinitis developing into asthma	49	11	37	21	10	34	35	10
Total	283	115	39	113	78	86=30%	129=46%	20

When asthma begins suddenly at about the age of 50, one hardly expects a large number of previous operations, but if sinal lesions are a part of the picture, one might expect many of them. They were found in almost half the cases. Thickened membranes in the sinuses and definite polypoid pansinusitis, often with retained secretion, were the two diagnoses, made in about equal numbers of cases. Most of the patients in both categories had polypi in the nose, and many polypi had been removed in the past, without benefit. Radical operations were done on 5 of these patients. Typical is the experience of a patient who was free of asthma for about six weeks after the operation and then had asthma again as badly as before. A year later he died in a typical attack of asthma, and autopsy showed his bronchi plugged with tenacious, inspissated material causing death by suffocation.

There were 4 deaths in this group. Two of them were those of patients in whom the nose and sinuses were apparently clear of gross lesions. One, already mentioned, had thickened membrane in both antrums; the fourth had true polypoid sinusitis, operation for which had been considered but was postponed when the attack became so much more severe.

In 2 patients the onset of severe asthma was attributed to an operation on the nose and throat; 1, a woman of 55 with chronic sinal trouble, first began to have asthma after polypi were removed; the other was a man of 52 with frequent colds and chronic sinusitis who dated his asthma from the time of a sinal operation. Both patients are still in trouble.

Finally, the group which is of particular interest comprises those who give a history of vasomotor rhinitis for several months before the onset of their asthma. Out of the total of 49 patients, 10 died of asthma. Permission for autopsy on 7 of them was obtained, and 4 showed typical bronchial plugs. The group includes about 17 per cent of all the cases of intrinsic asthma. Three fourths of the group were women, in whom the nasal symptoms began in their twenties and the asthma in their thirties. About 75 per cent of the patients had had previous operations on the nose or sinuses and, nevertheless, still had sinal lesions. On the other hand, a few remained clear of sinusitis, and it is important that even in spite of this freedom several died. One cannot say that the nasal lesion was the reason for the fatal outcome. In 3 cases, there was a history of bilateral radical antral operations done several years ago, and yet the asthma had continued, in 1 case to death from asthma at the age of 49. Cutaneous tests in the group were consistently negative, and many of the patients were admitted to the hospital ward without change in their symptoms. Thirty-one of the patients had both previous nasal operations and also present lesions in the nose and sinuses. Seven young women had had chronic nasal discharge, frequent obstruction and sneezing for many years—in 2 cases since childhood—when asthma began suddenly between the ages of 22 and 29. Three of the 7 women have died. There were 11 men and their stories were similar. The stuffy nose and frequent colds in the head had begun in the early twenties and then had led to asthma, which had continued persistently and increased steadily but surely in its severity despite all manner of treatment. Two of these men have died.

One was a young physician of 34 in whom a profuse watery nasal discharge with persistent obstruction developed, followed in two months by asthma, which continued intermittently for 10 years until his death at the age of 44. He had had two attacks of bronchopneumonia, each followed by temporary improvement in his asthma, and he had had two extensive radical operations on his antrums and ethmoid sinuses, likewise with temporary improvement but with no permanent gain. On several occasions, he had been admitted to the hospital when frequent doses of epinephrine were no longer able to control his asthma. He would vomit and fall into a virtual collapse. Twice, however, a sudden improvement in his asthma occurred without obvious cause, and the violent paroxysms ended by themselves as if by crisis. The last attack began like the others and progressed until he suffocated. Autopsy showed the lungs markedly distended and with the characteristic sticky plugs filling all the bronchi.

The other man had been troubled with symptoms referable to the sinuses for some months, and then, after an operation on his antrums asthma suddenly developed for the first time at the age of 30. From that time, asthma continued throughout all seasons, despite various admissions to the hospital, for thirteen years, until his death in a violent attack at the age of 43. He also was sensitive to acetylsalicylic acid. His treatment was always difficult and always unsatisfacfactory, although every known remedy was tried. His sinuses were exenterated; local treatments to the nose were given; bronchoscopic procedures were performed; his teeth had been extracted, and, finally, the thoracic chain of sympathetic ganglions was removed from his left side, with the development of a typical Horner syndrome but with no change in his symptoms. He died in the hospital ward, where he had been for several weeks, the last attack beginning in the same way as many other attacks. Autopsy showed the classic picture of distended emphysematous lungs, the cut section of which showed tough, tenacious plugs, which actually protruded above the cut surface.

There were 16 older women, each of whom presented a long history of chronic vasomotor rhinitis before the onset of asthma. Two of them died of asthma, although each had had several extensive operations on her sinuses. This group, with asthma after vasomotor rhinitis, is impressive, if only because 10 of the 49 patients have died. Whatever may be the relation between the earlier vasomotor symptoms and the later asthma, it is clear that operative removal of the sinal contents, including the membranes, had had no important effect on the

process as a whole. Many sinal operations have been done without change in the symptoms.

What to Do for Sinusitis in Asthma.—This report of our observations concerns a series of particularly difficult conditions, and it is all too evident that in them relief of asthma is unusual to say the least. However, if asthma of all kinds is taken together, improvement from surgical intervention occurs in at least half of the patients so treated, and this improvement may continue for weeks or months.

As pointed out by one of us (Weille, F. L.: Studies in Asthma: XVIII. The Surgical Treatment of Chronic Sinusitis in Asthma, *J. A. M. A.* **100**:241 [Jan. 28] 1933), the degree of improvement in the nose—the elimination of polypi, the removal of septal spurs and the control of sinal drainage—is not necessarily related to the degree of improvement in the asthma. Whereas the asthma may be relieved for months or years, it must be noted that the chances are nearly nine out of ten that sooner or later the wheeze will recur. These facts fit in well and provide strong support for the thesis that sinusitis is a part of the asthmatic syndrome and and not a cause of it.

Nevertheless, the disease in the upper respiratory tract can be reached and need not be neglected. A chronic postnasal discharge is a common cause of tracheobronchitis; it can make a bad matter worse, and if local treatment can modify it the patient should have the benefit of that local treatment.

As the rhinologist should be able to recognize allergy, so the allergist should be able to examine the nose. He can see the color and character of the nasal mucous membrane; he can note whether the septum is obstructive; he can recognize polypi, and he can examine the sinuses by transillumination. In short, he can obtain without much difficulty a rough idea of any nasal lesion which may be present, and so he can decide about the need for rhinologic consultation.

It is said that nasal disease should be treated on its own merits and without regard to the asthma. This is often true, but whenever "medical" and "allergic" treatment do not bring satisfactory results for the asthma within a reasonable time, the possibility of trouble in the nose should be considered before the asthma has advanced to a secondarily infected process demanding operation as a last resort.

What can the rhinologist do? His chief aim must be to relieve the postnasal discharge. He must prevent reflex disturbances from polypi, and he must consider focal infections. Meantime and most important, he must take great pains to disturb the normal structure and function of the nose as little as possible.

Shrinkage and suction, repeated at frequent intervals, with proper use of sprays between treatments, may accomplish much. In asthma the exudate often seen in the posterior pharyngeal wall has a dense, tenacious, sticky structure, comparable to that of the plugs seen in the bronchi at autopsy. Good control of the asthmatic paroxysm may sometimes result from its removal.

In many cases, it has been found that if local treatment to the nose can be continued systematically for a time, great symptomatic improvement of both the nose and the asthma may result. Recurrence of symptoms may be postponed until the true nature of the asthma can be found. Perhaps an extrinsic cause (dust) can be removed or some disturbance outside the respiratory tract can be treated so that the general condition of the patient can be built up enough to control the entire syndrome.

In other words, even if the nasal lesion is only part of the picture and even if local treatment to the nose will modify only a part of the patient's trouble, this modification may remove enough of the burden to allow a general improvement to occur.

SUMMARY

From a review of the literature as well as from personal observation of cases, the following findings are stated:

1. About a third of the new patients who apply for treatment of asthma have already had operations on the nose and/or sinuses, and about half have lesions at the time of examination.

2. The pathologic picture of the sinal lesions in asthma is characteristic and is similar to that of the lungs in asthma.

3. The results of further sinal operations leave much to be desired.

The thesis is proposed that asthma is only part of a syndrome which includes lesions of the nasal sinuses with formation of polypi and eosinophilia both in the lesions and in the blood stream and which depends fundamentally on an x factor so far unknown. Allergy is only one of the many exciting factors which in the presence of the x factor can precipitate symptoms.

If the nature of the sinal lesion indicates operation for itself without regard to the asthma, operative treatment will do good, but the good will be only partial and only temporary in most cases because the sinal lesion is a part of the picture and not a cause of it.

However, conservative local treatment may be worth while.

DISCUSSION

DR. W. J. MCNALLY: It is important for all otolaryngologists to realize the close association between allergic sensitivity and some diseases of the nose and throat, which Dr. Rackemann and Dr. Weille have stressed.

I fear that many of them are not fully aware of the work of Kern and Schenck, which strongly suggests that mucous nasal polypi are as much an indication of allergic sensitivity as is asthma or hay fever. Because of this association it is impossible to cure the patient by local nasal treatment unless the specific allergic irritant is determined and controlled. This is especially true of the more chronic nasal polyposis and sinusitis associated with bronchitis and asthma. The focus of infection in the sinuses can be eradicated completely only by an external operative approach to all the sinuses. Such a complete operation, however, is of little value unless the allergist is able to detect and control the allergic factors—the original cause of the whole cycle of the disease.

In addition to the closest cooperation between the allergist and the otolaryngologist, abundant patience and persistence are necessary to clarify this involved problem.

DR. J. S. L. BROWNE: I have been asked to discuss briefly the relation of histamine to allergy and anaphylaxis. I should like to associate Drs. S. Karady and B. Rose with these remarks. Manwaring was one of the first to suggest that anaphylactic shock is associated with a liberation of histamine in the dog. Code recently demonstrated clearly a marked increase in histamine in the plasma during anaphylactic shock in this species. The difficulty that histamine fails to cause the change in blood coagulation seen in this species has recently been obviated by the demonstration that heparin is liberated from the liver. Histamine liberated in the case of the guinea pig has been shown by Bartosch, Feldberg and Hagel to come from the lung itself. Karady showed that pretreatment with histamine would prevent anaphylactic shock in the guinea pig, and this has been confirmed by Smith. Karady and I have recently shown that histaminase given intravenously ten minutes before the shocking dose of antigen prevents anaphylactic shock in the guinea pig. Karady, Selye and Browne showed that mild nonspecific damage given twenty-four hours before the shocking dose ameliorates or prevents anaphylactic shock. In the case of allergic states, such as asthma, the effects of histamine pretreatment and histaminase have not as yet been clearcut, some authors claiming good effects and others failing to obtain them. One difficulty in using histaminase is that it is unstable and does not keep well except in tightly sealed ampules or capsules. Rose has recently demonstrated the release of histamine into the circulation after stimulation of the skin in cases of dermatographism and has also shown the importance of the adrenal cortex in the destruction of histamine. Roth and Horton showed that in sensitivity to cold histamine is liberated and that histaminase cures the condition. In connection with so-called physical allergy, Karady has recently advanced a new explanation. He has demonstrated that exposure of guinea pig serum or of the hindlegs of the guinea pigs to cold or heat produces changes in

the animals' own proteins which cause them to become antigenic; the substances thus produced he calls "auto-antigens." These scattered remarks may help to show the possible significance of the metabolism of histamine in allergic conditions.

DR. DAVID H. BALLON: I was impressed with the series of cases which Dr. Rackemann and Dr. Weille showed.

A few years ago I treated the condition in a series of cases with bronchoscopic procedure. As is known, bronchoscopic work on asthma has been done for about forty years. The early bronchoscopists injected cocaine and epinephrine, and it was evident that some of the excellent results obtained were due not only to these injections but also to the passing of the bronchoscope and the aspiration of the secretion. Some patients have associated bronchiectasis; others have a tenacious gluey secretion, so that in selected cases bronchoscopic procedure should be considered an aid.

As far back as 1925, I gave injections of iodized poppyseed oil to a number of asthmatic patients for diagnostic purposes, but in this procedure one must be extremely cautious. Only a small quantity of the oil should be used.

DR. H. E. MACDERMOT: Dr. Rackemann and Dr. Weille ventured into theoretic considerations this afternoon. May I ask them to embark on speculation and suggest what in their minds will be the description of the x factor?

The presence of an x factor in these strikingly severe conditions, which give physicians such hard work and such distress, is definitely to be reckoned with. In trying to handle such patients one comes continually to ask, "What can it be that is carrying on this chronic condition?"

DR. A. T. HENDERSON: Dr. Rackemann said that he was not going to say anything revolutionary or anything that was going to settle the whole question once and for all. I was rather relieved to hear him say that, because one is much more apt to take the things he said as understatement rather than overstatment. For a great many years Dr. Rackemann has stood for sanity and stability on the matters under discussion.

I want to congratulate the Section on Otolaryngology on their choice of Dr. Rackemann, and I feel that he has given all his hearers something sound and worth while.

The question ever arises of when to operate and when not to operate in such cases—how much to do and how much to leave undone.

One is likely to deal with antrums and hope that the patient will be better, perhaps forgetting for the moment that the ethmoid or other sinuses may be involved or that the lower respiratory tract may be the seat of infection.

A few cases have been observed here in which the ending has been fatal in which the same bronchial mucous plugs have been found. One case I recall, that of a woman who went into a status asthmaticus. She had an intrinsic type, which is difficult to deal with. She became unconscious and deeply cyanosed after a hypodermic injection of morphine. While she was still in coma, a roentgenogram of the chest was made, and it was observed that the diaphragm descended with inspiration and then recoiled violently into the chest. She figuratively had a rope tied around her neck, and she died from asphyxia. Even in the oxygen tent there was no relief.

One condition of this kind was relieved by intratracheal administration of ether. During the administration of the anesthetic, it was necessary to remove the catheter as the patient was not being anesthetized. The end of the catheter was found completely plugged with tenacious bronchial secretion, which had to be pulled out, and then satisfactory anesthesia and relief were obtained. Would bronchoscopic procedure in such cases be of value, or is the condition too critical to justify instrumentation?

DR. FREDERICK SMITH: I was asked by the committee to discuss what might be said about bacteria. I did not hear mention of a single organism, so that there is not a great deal for me to say.

I must confess that I am surprised that cultures of material from the sinuses are not made. As far as micro-organisms are concerned, one of the striking things is the variation in any particular case between the results of the cultures

made at different times during the course of the condition. In such instances it is difficult to determine which, if any, bacteria can be responsible. Nevertheless, there are conditions which seem to be related to the presence of micro-organisms in the respiratory tract. I remember one woman whose sputum yielded cultures of streptococci, and each great spasm corresponded with an increase in the organisms present, and lesser attacks corresponded with a decrease. She did remarkably well with vaccine from that organism. Two years later she had a relapse, but with no organisms present which fulfilled the criteria of the previous occasions. Vaccines at this time failed to help her at all.

As everybody knows, every sort of organism may be found in the sinuses. It is surprising that infections with some micro-organisms seem to respond to therapy and with others seem to do so badly. For instance, there is a fair regularity with which pneumococcic sinusitis responds to specific therapy and a marked irregularity in the response of staphylococcic infection.

Again, as far as bacteria are concerned, everybody realizes their complexity of structure, and it becomes an important matter to insure that the sensitizing antigen is present in the vaccine injected.

In lesions of staphylococcic origin part of the lesion is allergic sensitization of the tissues, and not infrequently active immunization is preceded by desensitization. A person may have relief with the first doses of an attempted immunization and frequently fail to benefit from subsequent courses given for relapse. The same holds true for other bacteria, and it is of more than academic interest to speculate on the relation of desensitization and hyperimmunization in attempting (1) to remove the sensitivity of the tissue and (2) to eliminate the focus of infection by the administration of vaccines and toxoids.

It is difficult to assess the current opinion on the value of vaccine in chronic asthmatic conditions, but I take it that there is a greater belief in its efficacy in Montreal than in many other places in North America. It does seem a striking thing that the speaker today failed to make any mention of bacteria, and I rather feel that my own opinion coincides with that of Dr. Rackemann on that point.

DR. RACKEMANN: I think I have speculated enough already. I do not know any more about this than the hearers do. There is no advantage in speculating too much—especially in public! One thinks of various things without getting very far.

As for bronchoscopic procedure, I think that it is a good point. Perhaps one would do better to use the bronchoscope before the patients get into such a desperate condition. One hesitates to do a bronchoscopic procedure, and by the time one gets around to it, it is often too late. Perhaps it would be wise to use bronchoscopic procedures sooner. The patients are not in too poor condition, because they are young people and their hearts are in good condition.

As for bacteria, I agree with Dr. Smith that cultures taken from the same patient at different intervals do not always show the same infection. The technic seems to be important. Some years ago I studied two series of cases, in one of which autogenous vaccines had been used and in the other stock vaccine. In these two series the results were similar. They were good only when the vaccine produced a local reaction at the site of inoculation. When the dose of vaccine is too large, malaise, fever and an increase in asthma may occur. We believe that the effect of vaccines is nonspecific.

There are five criteria of allergy:

1. Characteristic pathologic picture. (The symptoms of the patient must depend on this.)

2. Other manifestations of allergy (e. g., eczema, hives).

3. Family history. (If a man's son has eczema that is a sign supporting the diagnosis of allergy in the father.)

4. Positive cutaneous tests.

5. Eosinophilia.

Finally, as regards the taking of a history, I should stress the importance of using dates and accounting for all the time.

Book Reviews

Otolaryngology in General Practice. By Lyman G. Richards, M.D., Fellow in Surgery, Courses for Graduates, and Assistant in Surgery, Harvard Medical School; Associate Professor of Otolaryngology, Tufts Medical School; Research Associate in Otolaryngology, Children's Hospital, and Otolaryngological Surgeon, Peter Bent Brigham Hospital, Boston, with a foreword by D. Harold Walker, M.D., Professor Emeritus of Otology, Harvard Medical School; Past President, American Otological Society, and Former Chief and Present Consultant in Otology, Massachusetts Eye and Ear Infirmary, Boston. Price, $6. Pp. 352, illustrated. New York: The Macmillan Company, 1939.

The author, a distinguished otolaryngologist with a background of the finest traditions of this specialty, presents a textbook for the general practitioner. His chief aim has been to detail the essentials of otolaryngology in such a manner as to enable any practicing physician to gain the proper insight to handle a case intelligently and to seek help at once where his skill and talents may be limited.

This is not an easy task, especially when one recalls that about 20 per cent of general practice deals with diseases of the upper respiratory tract and that the advances in otolaryngology have been great in recent years.

The author has admirably succeeded in his purpose. He has compressed in 352 pages an enormous amount of information for the busy man who must have precise data for quick reference, in a literary style that makes for easy and interesting reading, omitting the more complicated anatomic, physiologic and pathologic discussions from the graduate student's point of view, avoiding as far as possible devoting too great space to the consideration of rare conditions, instead describing adequately the more common diseases of the ear, nose and throat by instruction in history taking, methods of examination and interpretation to the end that a definite conclusion is soon reached as to where the lesion may be and what therapy is indicated. The practitioner is guided carefully as to whether the method of treatment may be within his province or should be referred to the specialist.

This book should be in the hands of every general practitioner for frequent reference and review. It could be used with profit by medical students for collateral reading, by graduate students and by residents. Though the book may be somewhat terse and elementary for the specialist, the message of the author, because of his sound reasoning, wide teaching and clinical experience and surgical skill, merits the attention of his colleagues.

Medical Climatology: Climatic and Weather Influences in Health and Disease. By Clarence A. Mills, Ph.D., M.D., Professor of Experimental Medicine, University of Cincinnati. Price, $4.50. Pp. 296. Springfield, Ill.: Charles C. Thomas, Publisher.

The author stresses the importance of climatic environment to the basic physiologic reactions of the body, the combustion rate, energy level, rate of growth and development, resistance to infection and many other vital characteristics. He has assembled the facts from the knowledge thus gained, drawing freely from the researches of eminent workers, as well as his own first class contributions, into a volume of 296 pages.

The arrangement of the text and subject matter, the illustrations, type and format are excellent. Many diseased states are considered in the causation or perpetuation of which climatic environment may play a part. Some advice is given, in a number of the discussions, that should aid not a little toward the attainment of better results.

Colonization for climatically handicapped persons and the conditioning of indoor environments are detailed in separate chapters. The index is complete and informative.

Though some comments are made on keeping the extremities warm and comfortable during sudden climatic changes, it is hoped that the author will, in the next edition, enter more fully into the details of proper clothing to be worn during the varying seasonal changes in order to lessen the likelihood of chilling or overheating of the body. It is accepted by otolaryngologists the world over that climatic changes often excite or contribute to infections of the upper part of the respiratory tract.

This book is recommended to otolaryngologists interested in the relation of fluctuations of climate and weather to the upper respiratory tract, to research workers and, as a reference book, to physicians.

Eye, Ear, Nose and Throat Manual for Nurses. By Roy M. Parkinson, M.D., F.A.C.S., Head Oculist and Aurist to St. Joseph's Hospital, San Francisco. Fourth edition. Price, $2.25. Pp. 243, with 79 illustrations. St. Louis: The C. V. Mosby Company, 1939.

This is a textbook for nurses, prepared after a careful study of the needs for such a book and written in such a manner that it can be used to great advantage in teaching student nurses the things most important for them to know in order to carry out intelligently the specialist's orders and give the best possible care to their patients. The text is divided and outlined according to subject matter, so that it may readily be used for reference work as well as for general study. The language is not too technical, and those technical terms which it is necessary for the nurse to know are clearly explained. The illustrations, mostly done by the author and his wife, are excellent and help to clarify the descriptions of the anatomy and physiology of the parts dealt with and also to render the various operative setups and treatments easily visualized. Care is taken not to be too specific about details of treatment which may vary according to the individual preferences of specialists in each hospital, but the principles of treatment are emphasized so that students may understand the need for careful nursing care and the best ways to carry it out. One chapter deals with public health nursing and problems often met in the specialties concerned, and the information given therein can be useful to graduate nurses in all fields of work. Altogether, this book should be a valuable part of a nurse's library, whether used in class or merely to supplement her lectures.

Directory of Otolaryngologic Societies *

FOREIGN

Collegium Oto-Rhino-Laryngologicum Amicitiæ Sacrum

President: Dr. Louis Ledoux, Brussels, Belgium.
Secretary: Prof. Dr. C. E. Benjamins, Verlengde Heereweg 143, Groningen, Netherlands.

Hungarian Otolaryngological Society

President: Dr. V. Zimányi, Zárda-u. 48, Budapest II.
Secretary: Dr. G. Kelemen, Reáltanoda-u. Budapest IV.

Sociedad Rioplatense de Oto-Rhino-Laringología (Argentine Section)

President: Dr. Raul Becco, B. Mitre 1690, Buenos Aires.
Secretary: Dr. Juan Manuel Tato, Santa Fé 1171, Buenos Aires.

Société Française d'Oto-Rhino-Laryngologie

Secretary: Dr. Henri Flurin, 19 Avenue Mac-Mahon, Paris, 17e.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. A. W. Proetz, 3720 Washington Blvd., St. Louis.
Secretary: Dr. Leroy A. Schall, 270 Commonwealth Ave., Boston.
Place: New York. Time: June 10-14, 1940.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Frank Brawley, 30 N. Michigan Ave., Chicago.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.

American Bronchoscopic Society

President: Dr. Lyman Richards, 319 Longwood Ave., Boston.
Secretary: Dr. Paul H. Holinger, 1150 N. State St., Chicago.

American Laryngological Association

President: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.
Secretary: Dr. Charles J. Imperatori, 108 E. 38th St., New York.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Lee M. Hurd, 39 E. 50th St., New York.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Place: New York. Time: June 6-8, 1940.

SECTIONS:

Eastern.—Chairman: Dr. John R. Simpson, Medical Arts Bldg., Pittsburgh.
Place: Pittsburgh. Time: Jan. 5, 1940.
Southern.—Chairman: Dr. Walter J. Bristow, Doctors Bldg., Columbia, S. C.
Place: Columbia, S. C.. Time: Jan. 8-9, 1940.
Middle.—Chairman: Dr. Sam E. Roberts, Professional Bldg., Kansas City, Mo.
Place: Kansas City, Mo. Time: Jan. 19, 1940.
Western.—Chairman: Dr. Pierre Viole, 1930 Wilshire Blvd., Los Angeles.
Place: Los Angeles. Time: Jan. 26-27, 1940.

AMERICAN OTOLOGICAL SOCIETY

President: Dr. Horace Newhart, 527 Medical Arts Bldg., Minneapolis.
Secretary: Dr. Thomas J. Harris, 104 E. 40th St., New York.

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